THE
HEARTS OF MAN

R. M. WILSON, M.B.

OXFORD MEDICAL
PUBLICATIONS

Ex-libris
Charles Atwood
Kofoid
BMC

1868

THE LIBRARY
OF
THE UNIVERSITY
OF CALIFORNIA

PRESENTED BY
PROF. CHARLES A. KOFOID AND
MRS. PRUDENCE W. KOFOID

P 4/20

Jac. J. de Jong

THE HEARTS OF MAN

PUBLISHED BY THE JOINT COMMITTEE OF
HENRY FROWDE AND HODDER & STOUGHTON
AT THE OXFORD PRESS WAREHOUSE
FALCON SQUARE, LONDON, E.C.1

THE
HEARTS OF MAN

BY

R. M. WILSON, M.B.

LATE ASSISTANT TO SIR JAMES MACKENZIE, UNDER THE MEDICAL
RESEARCH COMMITTEE

LONDON
HENRY FROWDE HODDER & STOUGHTON
OXFORD UNIVERSITY PRESS WARWICK SQUARE, E.C.
1918

Anat

Printed in Great Britain
By Morrison & Gibb Ltd., Edinburgh

K-QP101
W8
Biol.
Lib

TO

MY FRIEND AND MASTER

SIR JAMES MACKENZIE

WHOSE GENIUS AND INSPIRATION HAVE OPENED THE
DOORS TO A NEW AND LIVING CONCEPTION OF MEDICINE

M354512

PREFACE

THE writer was so fortunate as to have his MS. read, at his request, by Sir James Mackenzie and Professor Bayliss. These two great authorities were kind enough to offer certain criticisms, and he feels that he will do his readers the best service if he quotes from these, and endeavours to explain his position in terms of answers to them.

Before doing this, however, he would like to explain the nature of the present book and the circumstances which caused it to be written. The prime object of the book is to encourage investigation into certain phenomena of the circulation and of the nervous system which have not hitherto been investigated from the purely clinical standpoint—for example, the relationship of the pulse to the respiration, the mechanism of breathing in effort and at rest, the meaning and effect upon the general circulation of the great "blood lakes" of the skin, abdomen, and lungs.

So little information was available regarding these matters that the author resolved to find out whether the study of the living individual in reaction and at rest would throw any light upon his darkness, and with this object in view observed various naked men and children during states of activity and relaxation. He was soon led to recognize two distinct and different types of breathing, and by the aid of Sir James Mackenzie's polygraph to make records of these. These records revealed to him waves in the abdomen and thorax which he was at a loss to understand, and very much labour was necessary before the explanations here given of them were arrived at. That these explanations are at variance with accepted theory makes further work on this matter by other observers necessary, for with every precaution and the best intentions it is difficult to avoid those miscalculations which strew the path of the clinical observer. The writer believes in his explanations: he is most anxious to avoid being dogmatic about them.

The earlier chapters are devoted to an account of the states of reaction and of rest as seen in many observations. The importance of this may not at first be evident to the reader, but if he perseveres with the text he will discover that the whole argument depends upon the ascertained pictures of gross physical reactions to the circumstances of life. He is therefore urged to read these early chapters with especial care.

A great many observations of a similar kind have been made upon patients suffering with diseases of various sorts. These are not included in the present work as they are not yet complete, and as they present a host of new problems which cannot well be dealt with in the small compass available.

The criticisms mentioned above are chiefly directed to the conception of a peristaltic wave in those arteries which join the blood lakes, *i.e.*, the arterial tree connecting the heart with the skin, and with the mesenteric blood lake. Sir James Mackenzie wrote :

" I scarcely grasp your meaning when you say there is a peristaltic wave passing down the arteries ; you give, for instance, illustrations of three waves, the anacrotic, the dicrotic, and the post-dicrotic waves. Do you mean to say that there are three waves passing down to one cardiac cycle ? The argument in favour of it is far from convincing. I remember going into the question of these waves, but my knowledge of physics was so imperfect, and I found those who knew more about physics were at loggerheads, that I gave up the attempt to explain the movements. But the anacrotic pulse is so well marked in cases of aortic stenosis that I think the explanation of that wave could be given without calling in the peristaltic wave."

In answer the writer would emphasize that he does mean that there may be three or more—or fewer—waves passing down the artery to every cardiac cycle. He regards the waves seen in a pulse tracing—always excepting the large "pulse wave"—as representing peristaltic waves in the vessel wall. This peristalsis of the vessel is an entirely different mechanism from the systole and diastole of the heart, and has no connection with it. But the systole of the heart by throwing blood into the arterial tree superimposes upon the peristaltic waves in the vessel another wave motion, the pulse.

On the other hand, there is evidence, as will be shown, that during the passage of the pulse wave the peristaltic waves are momentarily held in inhibition, while, during the diastole of the heart (*i.e.*, the inhibition of the pulse wave proper), the peristaltic waves are momentarily more active. This reciprocal mechanism is found also in the bowel between the sphincters of that structure and the areas lying between the sphincters. There is further reason to think—though this is merely mentioned by the way—that only the circular muscles of the vessels carry on peristalsis, and that during the period of the pulse wave, when peristalsis is momentarily inhibited, the longitudinal muscles go into contraction—an active inhibition of peristalsis being thus produced. In prolonged reaction states in young subjects a muscular spasm of the arteries is often observed and mistaken for arteriosclerosis. This may, perhaps, be explained in terms of active inhibition of peristalsis by contracture of the longitudinal muscles of the arterial wall.

As regards the anacrotic pulse in aortic stenosis, it is difficult to see how this disease condition can produce the waves observed, for though the narrowed orifice may cause the quantity of blood entering the aorta to be less for any given time period, nevertheless it is not likely to cause the entry to be in two successive "spurts." As the pulse wave is a form wave of the vessel wall, only by "spurts" could the wave be produced. Moreover, the wave is seen in cases with no aortic stenosis, and is not seen in other cases with aortic stenosis. It can be produced in certain healthy subjects by holding the hand above the head (a tracing showing this is given in the text).

Further evidence of the peristaltic condition in vessels is found in the changes seen in the pulse curve in various circumstances. These are dealt with in the text.

Sir James Mackenzie further dealt with the inspiratory wave observed on respiratory curves at each systole of the heart. He wrote :

"Your evidence of the systole of the thoracic veins by getting a record from the nose is also not convincing. These movements are very distinct, and were investigated, I think, somewhere in the late 'eighties by Delépine of Manchester, and his explanation is at all events the explanation that I have in my mind when I say that with each systole of the ventricle there is ejected out of the chest a certain quantity of blood, and the slight vacuum caused by this causes a slight inrush of air, and that is the reason of the characteristic wave that you got from the nose tracing. The same type of tracing can be obtained from other places, such as under the liver and from the sides of the chest, and I have got a number of records showing that undoubtedly the cause of this depression is the emptying of the heart of its blood."

To this it is necessary to reply that there is no question of a "systole of the thoracic veins." The conception is of a systole of the *lung arterioles* and possibly capillaries also—that is to say, of the whole pulmonary blood lake.

In the second place, it is worthy of note that the amount of blood which can produce a "vacuum" at each cardiac systole represents the difference only between the amount shot out of the chest into the abdomen, arms, head, etc., by the great arteries and the amount taken in by the great veins which are filling at systole—that is to say, it represents much less than half the contents of the left ventricle.

Yet this small amount of blood is sufficient to cause a vacuum capable of—

1. Causing a very appreciable intake of air by the nose (see tracings).

2. Causing a very appreciable inward sucking of the whole chest wall at the same moment.

3. Causing—by Sir James Mackenzie's showing —an indrawing "under the liver"; and by the writer's showing, a similar indrawing over the whole abdomen *into which the bulk of the arterial blood in question is being discharged.*

When viewed in this light, the idea that the vacuum created by the excess of the discharge of blood from the left ventricle over that coming into the great thoracic veins is the cause of these waves becomes in the highest degree improbable. On the other hand, if it be granted that the arterioles of the lungs go into systole and diastole with the heart, an immediate explanation of these waves is forthcoming. Further, a careful examination of the waves will show that not only is a downward curve observed in the breath tracing, an upward curve is also observed (see figures in text), the whole thing occurring during systole of the heart. Now, clearly, if the wave was due to a vacuum created by the exit of blood from the chest, this vacuum would reach its maximum at the *end of systole*. But the curve shows a maximum before the end of systole, and by the end of systole the "vacuum" is wholly filled up. This appears to the writer to be another argument against the vacuum explanation of the curve.

On the question of the peristalsis of arteries, Professor Bayliss writes:

"It is true that some evidence, of a very indirect and uncertain nature, has been brought in support of peristalsis in arteries, but it is very questionable and, I fear, will not be accepted as an explanation of the pulse curve. It is not necessary that a *closed* sphincter should be present to get a pulse wave; a mere resistance, such as that of the arterioles, is sufficient. The point is that the heart pumps in more blood at systole than can at once escape through the arterioles; it thereupon distends them, and the elastic reaction causes the pulse wave. It is clear that the blood expelled in systole possesses kinetic energy, and will continue to move after the semi-lunar valves are shut. This must produce some kind of a wave. It is not a question of a 'small reflection from the aortic valves.' The whole question is of great difficulty. . . . You may, perhaps, remember that Helmholtz himself was obliged to invent a new form of calculus to attack problems of the motion of liquids in tubes. . . . I feel sure that no treatment is adequate unless based on what causes the pulse wave, namely, the *elasticity* of the arterial wall. The description of the phenomena in terms of peristalsis conveys no meaning to me."

In reply it must be pointed out that while "a mere resistance, such as that of the arterioles," is quite enough to cause a pulse wave, relaxation of that resistance will, experimentally, always cause the wave to become smaller (which may be seen if the nozzle is removed from a Higginson's syringe). After the nozzle has been removed it will be much more difficult for the palpating finger to feel the stroke of the syringe bulb than before the nozzle was removed.

In the case of the pulse, however, as the tracings in this book show, when the arterial resistance is lowered (*i.e.*, when vaso-dilatation of the periphery occurs) the pulse wave becomes bigger—an exact opposite of the state of matters encountered experimentally. When, on the other hand, the arterioles contract and vaso-constriction of the periphery takes place the pulse wave gets smaller.

The mere conception of a fixed peripheral resistance will not cover this unless it can be shown that during dilatation the heart beats faster to keep the vessels full, and that during constriction it beats more slowly. Again, however, an examination of the tracings will show that the exact opposite occurs. During dilatation the heart beats more slowly, and during constriction the heart beats faster. It is therefore submitted that no explanation save that given will meet the facts.

Dealing with the vagus nerve, Professor Bayliss writes:

"The evidence that the vagus inhibits other vascular muscle than the heart is extremely weak. The depressor acts by reflex stimulation of all dilator nerves and inhibition of the tone of vaso-constriction centres. . . . I have not met with any evidence that the vagus acts on splanchnic or peripheral vascular areas. In fact, all attempts to detect it have met with failure."

The writer would reply that his meaning has been slightly misunderstood. He knows that the vagus nerve as such does not act upon the splanchnic or peripheral blood lake. But the depressor nerve does so and also acts upon the vagus. His conception was therefore what might be called a "vagus system," of which the nerve of this name was a mere fraction, playing but a part. This quarrel is thus really only about a name. It will be quite satisfactory to speak of the "dilator system," which is activated reflexly by the depressor nerve, and which includes the vagus nerve as one of its branches. The term "vagus sympathetic" has been used in the text only in this sense. Professor Bayliss' further observation on pituitarin is covered by the same explanation:

"Your argument about pituitarin is not convincing, because you assume that the depressor acts reflexly through the vagus, whereas experiment shows that it is through the various vasomotor nerves, and only the heart effect is abolished by cutting it."

The writer desires humbly to thank these great authorities for their kindness to him and for their criticism. That he has ventured to reply to them is due only to the fact that he has devoted much labour to the consideration of these problems.

system," which is nourished solely by the depressor nerve, and which includes the vagus nerve as one of its branches. The term "vagus system" has been used in the text only in this sense. Professor Cyon's further observation on phlorhizin is answered by the same explanation:

"Your argument about phlorhizin is not convincing, because you assume that the depressor acts solely through the vagus, whereas experiment shows that it is through the various vasomotor nerves, and only the least effect is abolished by cutting it."

The writer desires humbly to thank these great authorities for their kindness to him and for their criticism. That he has ventured to reply to them is due only to the fact that he has devoted much labour to the consideration of these problems.

CONTENTS

CHAPTER I

PAGE
REACTION BREATHING 1

CHAPTER II

THE FUNCTION OF THE ABDOMINAL WALL IN REACTION BREATHING 19

CHAPTER III

THE EFFECT OF THE THRUST OF THE ABDOMINAL MUSCLES UPON THE THORAX 29

CHAPTER IV

THE REACTION STATE AS AN ACCOMPANIMENT OF ALL EFFORT 37

CHAPTER V

THE ANATOMY OF THE ABDOMEN IN RELATION TO THE REACTION STATE 43

CHAPTER VI

THE EMPTYING OF THE ABDOMINAL BLOOD CISTERN . . 48

CHAPTER VII

REST BREATHING 56

CHAPTER VIII

PAGE

EVENTS IN THE CRANIUM DURING REACTION 63

CHAPTER IX

THE DUCTLESS GLANDS AND THE REACTION STATE . . 68

CHAPTER X

THE EFFECTS OF ADRENALIN 71

CHAPTER XI

THE RELATIONS OF THE BLOOD LAKES TO THE HEART . 85

CHAPTER XII

THE LAW OF THE INTESTINE 92

CHAPTER XIII

THE VAGUS—NERVE OF DIASTOLE 127

CHAPTER XIV

THE ACTION OF PITUITARIN 144

CHAPTER XV

THE RELATIONSHIP BETWEEN THE REST STATE AND THE REACTION STATE 161

CHAPTER XVI

ACUTE DISEASE AND THE NERVOUS SYSTEM . . . 170

INDEX 175

THE HEARTS OF MAN

CHAPTER I

REACTION BREATHING

THE familiar picture of action and the no less familiar picture of rest, as applied to the human body, have for long attracted the attention of lay writers who, by means of these pictures, seek to add conviction to their descriptions of emotions and events. They have not, however, attracted the attention of medical men to anything like the same extent. For some reason the medical man has been content to accept a dead rather than a living conception, a pathological rather than a physiological. He has been willing to study signs of disease with zeal and patience, but has overlooked the broad pictures of health with its manifold activities and reactions. He has missed the huge interplay of forces of which the resultant is a living organism, and so has failed to perceive that the difference between healthy life and diseased life is not fundamental but only quantitative. He has missed, too, the philosophical truth that there is no dividing line between health and disease any more than there is a dividing line between reaction and excessive reaction. Disease is ever functional, no matter what form it may assume; when it has ceased to be functional it has ceased to be disease, for the patient is dead. Health, too, is always measurable in degrees; if the degrees are few, health has given place to disease. Yet health also is only completely lost in death.

The Study of Simple Functions.—Knowledge of disease can therefore only be gained by knowledge of health, and that knowledge is at present exceedingly imperfect. It is most imperfect in respect of the simplest functions, because the very simplicity of these functions, the fact that all are familiar with them, has caused investigators to look elsewhere for their material. If, however, we are to obtain real knowledge, we must be prepared to begin at the very beginning, and must on no account refuse to study any function of life merely on the ground that it is so familiar as to be unworthy of notice.

One of the most commonplace reactions of the human organism is that seen when, by reason of a slight stimulus—for example, a fright of sudden and not very intense nature—the individual concerned recoils. This reaction is spoken of as "a start" in popular language. The writer has been at pains to study the reaction very closely in a large number of subjects over some three years. The following are the phenomena observed :—

The Phenomena of "Starting."—The patient instantly draws a sharp breath, which is of short duration and which enters his chest with a somewhat rushing sound, described popularly as a "gasp." At the same moment the upper part of the body is thrown backwards, pivoting, as it were, upon the lumbar vertebræ. The arms tend to be raised and to be drawn into a semiflexed position and the shoulders to be elevated. The mouth is sharply opened with a peculiar downward jerk which ends abruptly almost as soon as it has begun. The patient may become pale at this moment. There is also a tendency to gooseflesh. Following immediately upon the initial "gasp" the breath is held, *i.e.*, the glottis closes. The abdominal muscles become tense and the costal margins stand out prominently above a flat abdomen which at first is quite motionless. The eyes are fixed and perhaps even staring; the facial muscles grow stiff; the muscles of the neck, especially the sterno-mastoids are prominent, the lumbar curve of the spine is somewhat accentuated because the shoulders have been carried backwards. In the legs the gluteal muscles and the hamstrings stand out prominently.

If now the "start" continues, respiration is resumed. But the character of the respiration is not at all like that of quiet breathing. The abdomen scarcely moves at all, and the rigidity of the abdominal muscles is maintained so that the flat contour is scarcely altered. The muscles can be seen, in thin subjects, standing out. The costal margins remain very prominent and are seen to have been pulled away from one another in an outward and upward direction, as indicated by the arrows in Fig. 1. In this manner the chest has been opened without any appreciable increase in the size of the abdomen. During the act of expiration the tension of the abdominal

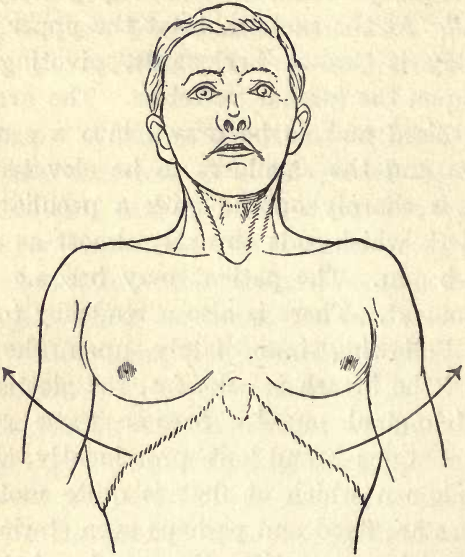


FIG. I.

muscles increases; expiration tends to be prolonged in a series of small explosive puffs as though the abdominal muscles were forcing the contained air against a closed glottis, which only at intervals gave way against them. In cases where the picture is exaggerated these explosive puffs produce a definite sound of a whimpering quality. Inspiration in succeeding breaths shows a short relaxation of the abdominal contour.

The Return to Normal after "Starting."—As soon as the occasion for excitement or alarm has passed the whole picture presented by the patient undergoes a change.

The tense condition of the abdominal wall is instantly relieved. The abdomen swells out to its usual contour, obliterating the prominence of the costal margins. The patient draws a long, deep breath, very different in type from the sharp, gasping, shallow breath of reaction. The abdominal wall moves out in front of this breath so that the curve of the abdomen is increased. At the same time the shoulders are allowed to move forwards, the arms hang limp, the knees tend to bend, the body assumes an easy, stooping aspect. The act of inspiration does not now appear to affect the relationship of the costal margins to each other to any marked extent, the forward movement of the abdominal wall allowing of free entry of air to the lungs. The act of expiration seems to be largely of the nature of an elastic recoil. The abdominal muscles do not become tense, they merely reassume their normal shape.

The Muscular Picture in "Starting."—The muscular picture presented during a "start" is worth attention. The most prominent features of it are the retraction of the head and its fixation in a more or less rigid position, the thrusting backwards of the thorax and its behaviour during respiration which have been alluded to above, the tension of the gluteal muscles and hamstrings causing a strong and fixed extension of the thighs, and the action of the hamstrings in holding the knee firmly at slight flexion. These actions, needless to say, are not pure actions of one group of muscles, but are general actions of the musculature emphasised in particular directions. The features may conveniently be discussed in some detail.

Retraction of the Head in "Starting."—This retraction is very well marked in certain of the cases, and is present in all when the stimulus provokes a decided reaction.

It is evidently due to the pull of muscles situated in the back of the neck, the trapezius splenius capites, etc.; but it is controlled by the action of muscles anteriorly situated so that whatever position of retraction the head assumes it is held rigid in that position. In fact a kind of balance on a fulcrum is produced, the fulcrum being the atlas vertebra. This is shown in Fig. 2.

The balance works in favour of the retracting muscles so that retraction will occur when the muscles come into simultaneous activity as occurs during the act of "starting."

The Effect of Retraction of the Head upon the Movements of the Thorax.—The observation that during the reaction of "starting" the two costal margins are pulled apart from one another so as to widen the angle between them, raises the question in what manner the thorax is thus forcibly opened.

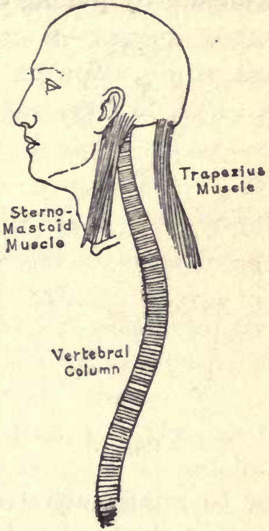


FIG. 2.

Clearly the action must pivot upon some fixed point situated above and behind the costal margins, for it is a matter of observation that the margins move outwards and upwards, giving the chest a wing-shaped appearance (Figs. 3 and 4). This point is evidently the spinal column directly; indirectly, as we shall see, other structures play a part.

The muscle which deserves attention in the first place is the trapezius. This muscle arises from the occipital bone (superior nuchal line and external occipital protuberance), the ligamentum nuchæ and the vertebral spines and intraspinous ligaments down to the last of the thoracic vertebræ. It is inserted into the outer aspect of the clavicle and the acromial process and spine of the scapula.

Its effect is thus either to retract the head by pulling against the shoulder or to retract the shoulder by pulling against the vertebral column or to raise and retract the shoulder by pulling against the head and vertebral column.

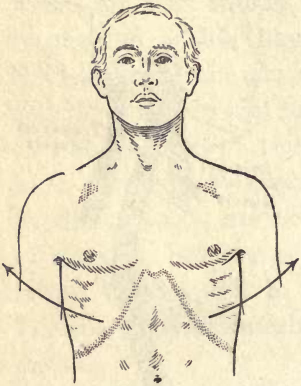


FIG. 3.

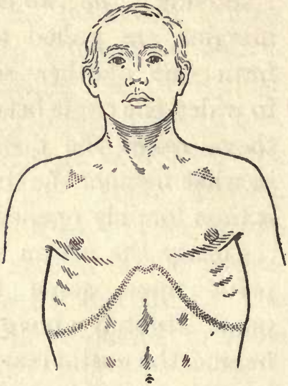


FIG. 4.

In point of fact, observation has shown that during the act of starting all these effects are produced, for the head is retracted, the shoulder is raised, and the scapulæ are brought closer together and stand out prominently in the patient's back.

The Action of the Sterno-Mastoid.—At the same moment the sterno-mastoid goes into active contraction. This muscle does not directly oppose the trapezius in the sense that it tends to pull the head forwards. The mastoid process is placed behind the atlanto-occipital articulation. If anything the sterno-mastoid would tend to pull the head back. In fact, careful observation shows that it really operates (the head being held retracted, and so fixed firmly on the fulcrum of the atlas) to raise the thorax. In other words, the muscle uses the fulcrum of the cervical vertebræ in order to pull up the clavicles and sternum and so raise the chest in front. The action of the sterno-mastoids is assisted by the actions of the scaleni muscles and the muscles of the front of the neck.

It is interesting to recall at this stage that the spinal accessory nerve supplies twigs to both the trapezius and the sterno-mastoid muscles.

The Pivot formed by the Scapula.—The head, therefore, thanks to this arrangement of muscles, forms during periods of sharp reaction, such as "starting," a pivot-point. It is held firmly in the most advantageous position by the trapezius muscle, and also, no doubt, by the other muscles at the back of the neck. Meanwhile the sterno-mastoid muscle bases its action upon the head, and by means of the pivot provided pulls up the top of the thorax.

Another pivot point has at the same moment sprung into existence in the shape of the scapula. As we have observed, the action of the trapezius muscle drew the scapulæ inwards towards the vertebral column, causing a hollow to appear between the two blades. At the same time the upper fibres of the trapezius in retracting the head also tended to raise the scapulæ.

The scapulæ thus, as may be observed easily, are firmly held towards the backbone and in a slightly upward direction; and in this action, in addition to the trapezius, the rhomboid muscles, the levator anguli scapulæ muscle, and others evidently take part.

The scapula becomes by this means a fixed and pivot point just as the head becomes a fixed and pivot point.

Acting upon this pivot point is the serratus magnus muscle which by using the scapula in the manner in which the sterno-mastoid uses the head is able to raise the ribs and at the same time pulls them outwards. This is shown in transverse section in Fig. 5.

The Shoulder an Additional Pivot.—Further, the action of the latissimus dorsi at this moment is to pull the shoulder backwards, and by so doing to force up the point of the shoulder by drawing the head of the humerus inwards against the glenoid fossa. This action is supported by that of the pectoralis major, which can also be seen to tighten during the reaction of "starting." It is further supported by that of the deltoid, which always contracts at this moment, and which accounts for the raising of the arms already referred to.

The shoulder pivot thus directly supports the scapular pivot, and so renders the pull of the serratus magnus muscle the more effective.

From the shoulder pivot the pectoralis minor muscle acts, assisting the serratus magnus in raising the ribs and pulling them outwards.

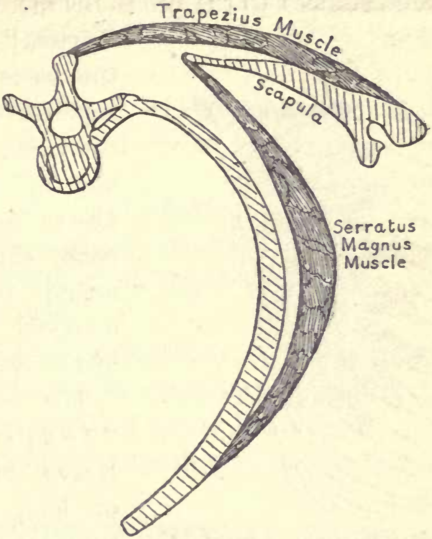


FIG. 5.

The Action of the Serratus Magnus Muscle on the Costal Margins.—The serratus magnus "arises" from the upper eight or nine ribs on their antero-lateral aspects, and is inserted (Fig. 6, after Cunningham) into the costal aspect of the vertebral border of the scapula. This origin and insertion, granted that the scapula is a fixed point and is well pulled back towards the vertebral column, makes it a matter of certainty that the muscle in contraction must pull asunder the costal margins.

An action of a similar kind clearly belongs in the same circumstances to the pectoralis minor, which "arises" from the surfaces and superior borders of the third, fourth, and fifth ribs near their anterior ends, and is inserted into the coracoid process of the scapula.

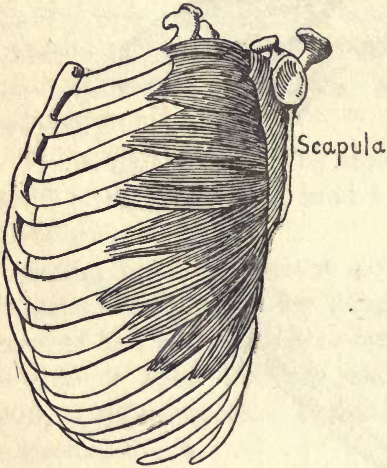


FIG. 6.

The so-called "Inspiratory Nerve of Bell" or long thoracic supplies the serratus magnus.

The Mechanism of Reaction Breathing. — The mechanism of reaction breathing evidently depends on two primary fixed pivot points — the head—retraction of which allows of the clavicles and sternum being pulled up; and the vertebral column—the use of which by the trapezius muscle allows of the scapula being held in position. From the secondary fixed points thus created, the elevated clavicles and sternum, the fixed scapula and the raised shoulder, another set of muscles act to open the chest and cause inspiration. These muscles are :—

Acting from the elevated sternum and clavicle	The external intercostals and the internal intercostals.
Acting from the fixed scapula and raised shoulder	The serratus magnus and pectoralis minor.

The mechanism of reaction breathing is therefore divisible into two parts :—

1. The mechanism which produces the secondary pivot points—consisting of the trapezius muscle, the sterno-mastoid muscle, the pectoralis major, the latissimus dorsi, the deltoid and many of the deeper muscles.

2. The mechanism which produces actual inspiration by opening the chest and separating the costal margins—consisting of the intercostal muscles, the serratus magnus muscle, and the pectoralis minor muscle.

The Mechanism of Expiration in Reaction Breathing.—Observation has shown that not only inspiration occurs in the reaction type of breathing: expiration also occurs. As we have already seen, the expiration, like the inspiration, belongs strictly to the picture, and is quite unlike the expiration seen in quiet or resting breathing. The abdomen remains flat and contracted, it changes its contours scarcely at all. All that is noticeable is a tightening of the recti muscles and of the other muscles of the wall, and, in the epigastric region, a deepening of the sulcus between the costal margins.

Applying this observation, we conclude that the secondary pivot points (the elevated clavicles and sternum, the fixed scapulæ, and the raised shoulders) remain a feature of expiration in reaction breathing just as they were a feature of inspiration in this breathing type. It is easy to demonstrate this truth, and it has been proved by many observations.

Moreover, the separated costal margins do not to any marked extent approach one another again as might perhaps be expected, but, on the contrary, remain separated, and so, as we shall see, constitute a third fixed pivot point in relation to the act of expiration during the reaction type of breathing.

The Costal Margins as a Pivot Point of Expiration.—Springing from the ribs by a series of interdigitations with the serratus magnus muscle is the external oblique muscle of the abdomen. Springing from the costal margins, anteriorly, are the recti muscles. These muscles are reinforced by the internal oblique and the transversalis abdominis.

The action of these muscles is—if the pelvis be taken as a fixed point (or if it be assumed that the active gluteals will prevent the pelvis from being drawn up towards the sternum)—to draw down and in the costal margins and to flatten the abdominal wall upon its contents. If, however, the costal margins cannot be drawn down, owing to the strong action of the muscles holding them up and apart, and if the whole thorax cannot be pulled forwards upon the pivot of the lumbar vertebræ (owing to the strong action of the erector spinæ muscles which are always tense during the reaction of "starting"), then the effect of the contraction of the abdominal group of muscles will be to compress the abdominal viscera upwards into the space under the diaphragm, and thus to compress the thoracic viscera also and to force air out of the lungs.

In this act of compression the separated costal margins will form a pivot point so long as they do not yield to the downward pull of the muscles acting from them.

Inspiration following Expiration in Reaction Breathing.—It is manifest that if now the picture of reaction be maintained and the costal margins continue to be held apart by the serratus magnus muscles and the other muscles performing this function, inspiration of a shallow type will occur the moment the squeeze of the abdominal muscles is released, and expiration will occur the moment it is restored. This inspiration will take place because the pressure exercised from below upon the thoracic contents will be relieved as the abdominal contents slip down, and this expiration will take place because the pressure exercised from below upon the thoracic contents will be increased as the abdominal contents are thrust upwards by the contracting muscles of the abdominal wall.

Differentiation of the Gasp which occurs at the outset of Reaction from the Inspiratory Phases occurring after the Reaction Picture has been established.—A careful study of the living subject reveals the fact that the initial gasping sound heard when the stimulus is applied and the patient startled occurs at the moment when the costal margins are forcibly pulled asunder by the serratus magnus muscles, and when the whole chest is opened and pulled up by the other muscles of this group. The gasp, therefore, is due to the sudden inrush of air into the lungs which occurs when the chest is thus forcibly distended. The size and shape of the chest does not, after this, and while reaction continues at the same high level, undergo alteration. The costal margins do not to any marked extent approach one another again, the activity of the muscles holding them apart is not relaxed. From this point onwards expiration and inspiration in so far as they are performed are carried out by the abdominal muscles. At each squeeze of these muscles an expiration takes place and at each relaxation an inspiration.

The gasp, therefore, is a phenomenon apart and belongs to the initiation of the reaction type of breathing. It is not repeated except in response to a subsequent stimulus.

The Extent of the Muscular Mechanism employed in Reaction Breathing.—It is easy to demonstrate the fact that reaction breathing, at least in its well-defined manifestations, calls into play all the muscles of the trunk and limbs, in fact all the voluntary muscles with a single conspicuous exception.

This is evident when the picture of a startled man or animal is called to mind. In man the leg muscles play an important part in rendering the pelvic brim a fixed point, the back muscles perform a similar function in preventing the costal margins from being pulled towards the pelvis, the muscles of the arm assist in fixing the shoulder and producing that rigidity which is essential to the action of the muscles holding the scapula in its proper position.

The muscles of the mouth and alæ nasi also act, affording a freer passage for the inrushes of air.

The Diaphragm is inhibited during Reaction Breathing.—It is clearly not possible to make immediate observations upon the diaphragm in living subjects who are in active movement. Continuous observation, however, of these subjects can, we think, lead to no other conclusion than that, during active reaction, this muscle is held in inhibition.

Certainly the diaphragm plays no part in the original gasping inspiration with which the reaction state is ushered in. This is evident from the pulling apart of the costal margins, an event entirely contrary to the effect of a diaphragmatic contraction, which would tend to hold the costal margins in closer apposition. Indeed, unless the diaphragm be inhibited at this moment, it is difficult to see how the picture can be produced at all, for the abdomen is tense and flat and does not move out while the air is entering the chest. It moves inwards, on the contrary, to assume the tense and flat aspect. The abdominal viscera are pushed upwards in front of the abdominal wall during this inward thrust, an event which could not possibly take place if the diaphragm was actively descending at the same moment. During quiet or resting breathing, on the other hand, the diaphragm, unaided, carries on the process of respiration.

CHAPTER II

THE FUNCTION OF THE ABDOMINAL WALL IN REACTION BREATHING

IF the foregoing statement of observations made has been sufficiently explicit it will be evident that in reaction breathing in its pure form the muscles of the abdominal wall play an extremely important part.

In the first place, during the preliminary gasp, while the reaction state is being ushered in, these muscles are flattened out on the abdominal contents by the sharp upraising of the thorax and costal margins. This is not a mere mechanical stretching, for the rigidity of the muscles is increased though they do not go into powerful contraction. It amounts indeed to an increase of tone of the whole abdominal musculature. Following this, as we have seen, the abdominal muscles contract powerfully, forcing the viscera up against the thoracic viscera and so expelling air from the chest. If at this moment the glottis be closed the thoracic and abdominal viscera will be powerfully compressed. It was observed in reaction that the glottis did tend to close against this expiratory effort of the abdominal muscles, and that the breath was expelled as a rule in a series of short puffs occasioned by the opening and closing of the glottis while the tension of the abdominal muscles was maintained.

Each time that the contraction of the abdominal muscles was relaxed, air entered the chest in inspiration. This relaxation of contraction, while the reaction state continued, did not mean a relaxation of tone; the increased tone of the abdominal muscles was kept up even when those muscles passed out of the state of active contraction.

In the Reaction State the Expiratory Phase is the Active Respiratory Phase. — It is clear, therefore, that, in the reaction state, once this state has been established, the expiratory phase and not the inspiratory phase is the active phase of respiration. The strong and active contraction of the abdominal muscles, often against a narrowed glottis, is expiratory in effect; the partial relaxation of the abdominal muscles which follows this contraction is inspiratory in effect. Expiration represents effort, often very powerful effort; inspiration represents the interval intervening between these expiratory efforts.

The Reaction State affords the Abdominal Musculature full opportunity of emptying the Thorax.— This brings us to the point of view that the whole picture of reaction breathing represents a posturing of the body so that the abdominal wall, in its forceful contraction, may be enabled to exercise pressure upon the thorax from below, and so be enabled to expel air or other content from the thorax. The truth of this can again be confirmed by examining the living subject. Coincidently with the pulling up and out of the thorax in the reaction state, we may observe, as has been described, a drawing up, in, and backwards of the shoulders and a retraction of the head. The effect of this drawing up, in, and backwards of the shoulders in which the pectoralis major muscle, the latissimus dorsi muscle, and the deltoid muscle take part is to bind the thorax at the top while it is opened below. The shoulder girdle is rigid and raised. It is held firmly, affording a strong pivot-point for the arm. The pectoralis major muscle is drawn out flat on the top of the thorax in this process and tends to hold the top of the thorax in front. Behind, the top of the thorax is held by the encircling belly of the serratus magnus as it sweeps up to the scapula.

The effect is to render the chest more triangular in outline, so that we have a broader inlet below from the abdomen and a narrower outlet above into the neck. In other words, a somewhat "barrel" shape is changed into a somewhat "funnel" shape as in Figs. 7 and 8. Necessarily any pressure acting at the arrow point produces a more sudden and more intense effect at the point x in the "funnel" shaped chest than in the "barrel" shaped chest.

Indeed the "funnel" shape assumed by the chest in the reaction state corresponds to a syringe of which the diaphragm—in a state of inhibition and relaxation—forms the "ball," the trachea and large blood vessels the "nozzle." The abdominal wall is the hand of the operator compressing the "ball" of the syringe. The idea is illustrated diagrammatically in Fig. 9.

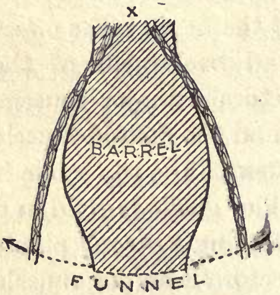


FIG. 7.

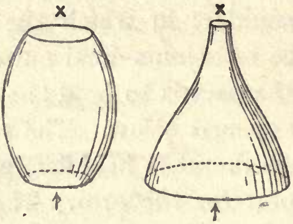


FIG. 8.

The Abdominal Viscera form a Pad which acts like the Piston of a Syringe.—Between the abdominal wall and the diaphragm there are interposed the great abdominal viscera which must necessarily bear the brunt of the squeeze given by the abdominal muscles, and which, thus, act in a passive way, like the piston of a syringe. There can be no doubt that the abdominal viscera are well suited to the performance of this function.

In Quain's *Anatomy* an impressive description is afforded of the contents of the abdominal and pelvic cavities viewed *en masse*. (Quain, 11th ed. 1914, vol. II. Part II. p. 68 *et seq.*)

"The upper and larger portion of the mass occupying the abdomino-pelvic cavity has its longest axis vertical, and under ordinary conditions its transverse diameter considerably exceeds its antero-posterior. . . . The upper end of the mass is convex and closely applied to the concave under surface of the diaphragm, and it must necessarily vary in form with the respiratory movements of this muscle. The anterior aspect

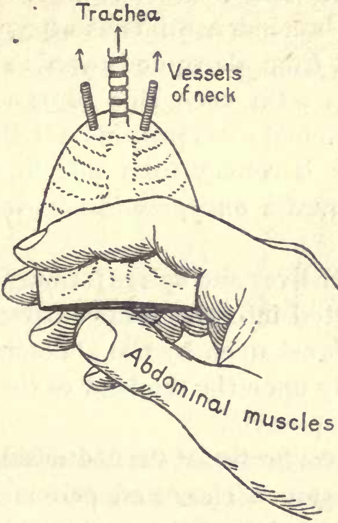


FIG. 9.

may be concave, flat or convex, according to the degree of retraction or protrusion of the anterior abdominal wall. *The posterior aspect of the mass has a more constant and characteristic appearance than the anterior. A deep median groove extends from above downwards as far as the junction of the upper and lower portions of the abdomino-pelvic cavity,* where it becomes continuous with a surface which is convex from above downwards and from side to side. . . . On each side of the median groove the abdominal mass presents a well-marked eminence, which is convex from side to side, *but diminishes in breadth and prominence from above downwards.*"

This mass, with liver and spleen forming its solid substance, is fitted into the syringe-barrel of the thorax and is thrust upon by the abdominal wall. It strikes directly upon the tambour of the relaxed and inhibited diaphragm.

The Manner of Contraction of the Abdominal Muscles.—If we would gain a clear and permanent conception of this syringe-action, we must closely and carefully study the method of contraction of the abdominal muscles in the living and active subject.

It will be found that in reaction states the abdominal muscles always carry out their contraction from below upwards, and in a wave-like motion. A diagrammatic picture of this is shown in Fig. 10.

Clearly enough this upward wave of contraction (we shall for the moment neglect the effect of the wave on the pelvis) will act just as the finger acts in pressing the piston of a syringe home. The wave of contraction will sweep the abdominal contents up under the ribs and against the diaphragm and hold them there. Air and blood will be powerfully syringed out of the thorax.

The Anatomy of the Abdominal Wall suggests its Syringe-like Action.—It is to be observed that the muscles of the abdominal wall are arranged in such a manner that the entire chamber can be squeezed in every direction except at the top where the costal margins are interposed; they are also so arranged that the contents of the chamber can be thrust upwards into the area under the ribs (and downwards, in the case of the lower viscera, into the pelvis).

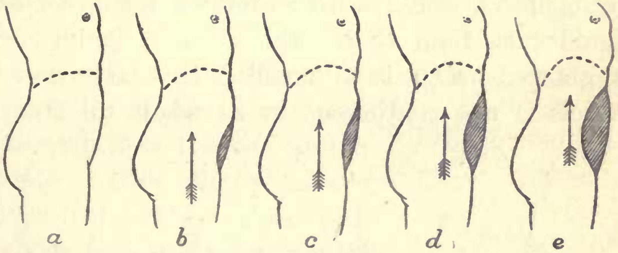


FIG. 10.

There is first of all a circular sheet of muscle, the transversalis abdominis; then there are oblique muscles running across one another; finally, there are longitudinal muscles extending across the cavity in a vertical direction.

Each of these muscles, in a different way, act to force the viscera against the diaphragm. Their actions are correlated in the mechanism of the sheath of the rectus.

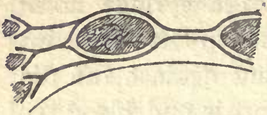


FIG. 11.

This sheath (Fig. 11) is so constituted that the various muscles can work as one muscle, but the arrangement would seem to have a much deeper significance than that. This deeper significance is grasped when it is recalled that the rectus muscle is not continuous in its whole thickness, but shows three or more *tendinous intersections which are adherent to the sheath of the muscle.* In other words, if these attachments of the rectus to its sheath should become fixed points the muscle would be able to contract between them. The importance of this becomes apparent when the

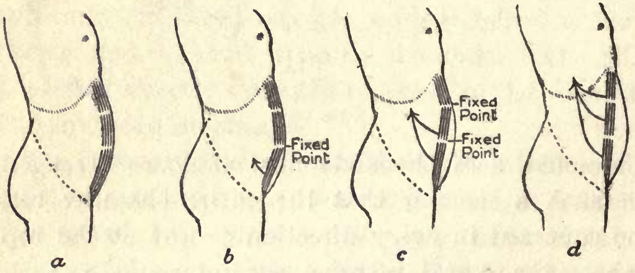


FIG. 12.

abdomen is observed in the reaction state and the "wave of contraction" which sweeps over it from below upwards is noted.

In Fig. 12 it is made clear how those fixed points formed by the attachments of the intersections to the sheath allow the muscle to contract in separate pieces, and so produce the upward rolling or wave movement which can be observed.

What the diagram does not show is the effect of the oblique and transversalis muscles which by their contractions produce the fixed points. These muscles gird the abdomen, drawing it in, and this especially in the lower part where all their tendons pass in front of the rectus muscle (Fig. 13). They thus pull against each other and render the rectus sheath absolutely firm and fixed. Then the rectus, as can be seen, begins to contract in pieces, the lower portion between the pubes and the first intersection first, and afterwards, in order, the upper portions.

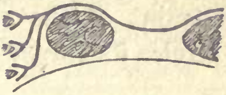


FIG. 13.

Thus not only does the space surrounded by the ribs form a barrel for the syringe of which the viscera are the piston; by the action of the encircling abdominal muscles the barrel of the syringe is continued right down to the pelvic brim. It then only remains for the expelling force to begin its work at the pelvic brim (rectus muscle), and to thrust the piston (viscera) up through the length of the barrel.

In Fig. 14 a diagrammatic representation of this thrusting action of the rectus muscle is given.

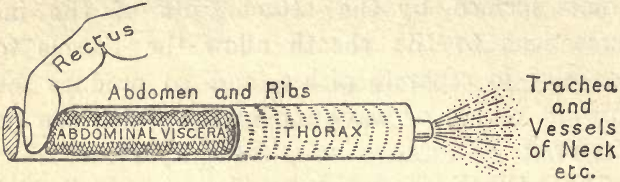


FIG. 14.

Below the folds of Douglas, *i.e.*, in the lowest part of the abdomen, the rectus, free behind from the restraint of the sheath, will clearly be able to exercise its thrust most effectually and thus begin the movement of the plunger with the greatest possible advantage.

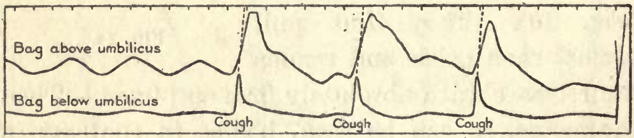


FIG. 15.

Note how the wave of contraction begins below the umbilicus and spreads up.

In Fig. 15 the fact of the upward character of the wave of contraction in the recti muscles is shown clearly in a polygraph tracing.

CHAPTER III

THE EFFECT OF THE THRUST OF THE ABDOMINAL MUSCLES UPON THE THORAX

GENERALLY speaking it is obvious that the thrust of the recti muscles must tend to empty the thorax just as the pushing in of its piston tends to empty a syringe. Moreover, as we saw, owing to the shape assumed by the thorax during reaction breathing, this thrust by the recti muscles takes place in conditions of advantage, the chief of which is the narrowing of the " nozzle " of the syringe in the shape of the upper part of the chest. The contents of the thorax might therefore be expected to leave it with very considerable force.

The Contents of the Thorax.—The main movable contents of the thorax are air and blood. Now it is a curious point of observation that just at the moment when the thrust of the recti muscles is taking place the glottis tends to shut and so prevent the air content of the thorax from being driven out. This is an observation about which no sort of doubt exists, and it can be made by any one who cares to induce the active reaction state in another.

But if the air is prevented from leaving the thorax at this moment then blood must leave the thorax in order to satisfy the strong demand of the contracting abdominal muscles.

In point of fact it is easy to demonstrate that this is exactly what occurs. One has only to watch the great vessels of the neck in order to be convinced.

During the abdominal thrust blood leaves the thorax by every available channel.

This again is a matter of observation. The neck can be seen to throb ; the blood pressure in the arms and legs is raised, the blood can be felt by the patient surging into the face and limbs if the process is an active one. Moreover, on merely physical grounds this effect must follow this cause because the pressure induced by the piston must find relief in some direction, and can find relief in no other direction so long as the glottis remains closed. The thrust of the recti muscles in no way acts as an impediment to a free outflow of blood by the abdominal aorta, but rather tends to encourage such outflow by producing a measure of suction behind the rolling wave of the muscular contraction.

The output of air from the glottis acts as a regulator of the output of blood from the thorax.

This again is manifest so soon as the meaning of the short, puffing expiration (like a series of short, small coughs) which is characteristic of the reaction state is understood. Each time the glottis opens and air is expelled the amount of blood expelled is reduced; the air expulsion eases the pressure acting upon the thorax and so reduces the necessity for blood expulsion. On the contrary, the less often the glottis is opened the more force will be available for driving blood out of the thorax. Further, the fuller the thorax happens to be with air at the moment of the abdominal thrust the more effective will that abdominal thrust prove in driving blood out of the thorax.

The air in the thorax plays the part of a pneumatic buffer between the blood and the abdominal wall.

This pneumatic buffer acts in two ways apparently :—

1. It affords a protection to the lungs while the blood is being squeezed out of them.
2. It diffuses the thrust of the abdominal muscles according to the laws of the expansion of gases.

It protects the lungs because the air pressure can be regulated by the glottis so as to afford the required degree of tension and so as to relieve excess of tension. It assists in the emptying of the lungs by securing an even, expelling effect upon the blood channels.

This will be more evident when it is recalled that the great mass of the blood in the thorax at any given moment is situated in the lungs. If effective emptying of the thorax is to be carried out—the emptying must come from the lungs principally. The contents of the lungs are air and blood, and air is not driven out because of the closure of the glottis.

The Manner in which Blood is driven out from the Lungs in Reaction States.—The diagrammatic figure, Fig. 16, gives an idea of the manner in which blood is expelled from the lungs under the thrust of the abdominal muscles. The lung capillaries, in fact, are squeezed by the air in the alveoli and so emptied. The whole lung becomes relatively more full of air and less full of blood.

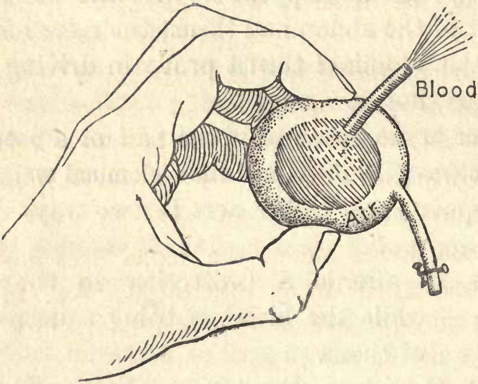


FIG. 16.

Oxygenation of Blood is stopped or diminished during the Reaction State.—It necessarily follows that during the reaction state the process of oxygenation of blood and discharge of CO_2 from the blood is greatly reduced or comes to a standstill. No other conclusion is possible. It follows, therefore, as a further conclusion, that during reaction states the waste-product content of the blood must rise rapidly—or to put the matter more explicitly—*must be forced to rise rapidly.*

The Effect of emptying the Lungs of Blood is to engorge the Arteries with Blood.—This again is an inevitable conclusion from the premises. The blood contained in the lungs must, on being driven out of these organs, go to swell the amount of blood in the arterial tree. This effect may be proved by a series of blood pressure readings on the brachial or other systemic artery.

The Blood driven out of the Lungs does not pass to the Abdominal Blood Cistern nor yet to the Peripheral Blood Cistern.—The fact that the blood driven out of the lungs does not pass into the great abdominal blood cistern, formed by the mesenteric vessels, is self-evident. At the very moment when the blood is leaving the lungs the abdominal blood cistern is being subjected to exactly the same pressure as that exercised upon the pulmonary blood cistern. The bowels, under the thrust of the recti muscles, are being heaped under the costal margins and squeezed against the liver and spleen. They are indeed being used as a part of the " piston " of the syringe to which reference has already been made.

It is interesting to note here in passing that the air in the bowels probably serves here again as a pneumatic buffer between the thrust of the recti muscles and the mesenteric vessels. The blood from the lungs, therefore, can find no lodgment in the mesenteric vessels. On the contrary, the blood from the mesenteric vessels is undoubtedly at this moment surging through the portal system to find a lodgment for itself. This matter will be discussed later. Nor can the blood from the lungs find a lodgment in the peripheral blood cistern, for, as observation shows, the onset of the reaction state—in marked cases—is associated with pallor of the skin which may be very intense and which is always more or less well marked if the reaction state is an active one.

This pallor is in marked contrast to the throbbing of the great vessels in the neck. It may be accompanied by sensations of cold in the extremities and indeed all over the body—" cold shivers." There is thus reason to suppose that far from affording a lodgment for blood from the lungs, the peripheral blood cistern is in itself being emptied at this moment.

There are only two possible situations into which blood discharged from the Pulmonary Blood Cistern and other blood cisterns during the Reaction State can go—the Muscles and Cerebral Cavity.—The blood, as we have seen, cannot enter the abdominal blood cistern, nor can it enter the peripheral blood cistern.

It must, therefore, since there is no other place for it to go, and since it certainly goes somewhere—enter the muscles and cerebral cavity. In the huge muscular field of the body alone is there room for it. The increased muscular and mental activity which, as we have seen, accompanies and is a part of the reaction state is by no means inconsistent with this view.

The same forces which operate to empty the lungs of blood must also operate to empty the heart in its systole and to refill it rapidly in its diastole while the rush of blood from the lungs continues.—In order to escape from the thorax the blood from the pulmonary cistern has to pass through the left heart. It is coming to the left heart under increased pressure, and will therefore fill that heart with force during diastole. During systole the force of the ventricular muscle will be augmented by the pressure acting upon the muscle from without, *i.e.*, the pressure exercised on the cavity by the recti muscles.

This pressure will necessarily act also during diastole, but the force of the blood rushing from the lungs is so much more conspicuous that the pressure here is probably negligible. The capillary lake of the lungs opening out into the narrow pulmonary veins affords clearly another example of the syringe and nozzle mechanism (Fig. 17).

It is a matter of universal observation that the heart is accelerated during the reaction state, and that its beat is also augmented in force, as revealed by the apex thumping the chest wall.

In the case of the right heart the same condition will be met with. This we shall discuss under the heading "The Emptying of the Abdominal Blood Cistern."

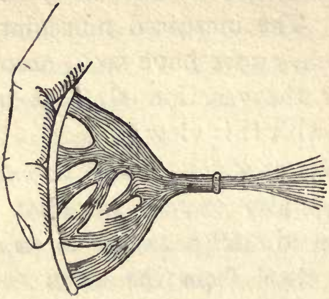


FIG. 17.

CHAPTER IV

THE REACTION STATE AS AN ACCOMPANIMENT OF ALL EFFORT

WE have so far been considering the reaction state in what may be called its "pure" form. It is manifest, however, that the ordinary reactions of life are not of this extreme kind. It therefore becomes a matter of importance to determine whether or not these lesser reactions partake of the nature of the greater reactions.

With the object of determining this matter a large number of observations were carried out during all kinds of action states and excitement states. It may be laid down at once that the result of these observations and experiments was to make it quite clear that all reactions and activities, whether of the body or the mind, demand for their accomplishment a greater or less degree of what we have called the "reaction state," and described as such above.

Every time that close Perception or Active Mental Effort is engaged in, the Glottis and the Abdominal Muscles show an increased tone.—This observation may be carried out by any one on himself.

If, for example, the attention is focused to hear a distant sound, the glottis will be closed and a light degree of increased abdominal tone will occur. The effect, as we have seen, is to retard blood return to the thorax and empty blood out of that chamber. The immediate result of this must be to increase up to the necessary limit the blood-content of the brain. The effort is not enough to accomplish more than this—indeed it is so slight an effort as, generally, to escape attention. *But it is essentially the same effort as that we have been describing, and it is produced by essentially the same mechanism,* as can be seen when the patient lies naked. The same thing exactly occurs when an attempt is made to focus vision closely, or to think closely. The following polygraph tracings made from healthy people illustrate this matter.

In Fig. 18, which represents nasal respiration taken by a tube in the nostril connected with a polygraph, it is seen that on the order being given to " listen intently " the patient " held his breath." He did the same thing on being told to look intently at a distant object (Fig. 19). At the same moment the abdominal muscles were observed to move slightly, becoming a little more tonic. Fig. 20 shows both events—looking and listening; and Fig. 21 shows the muscular effect on the abdomen.

The Effort of Speaking.—The effort of speaking affords a good illustration of the "closed-glottis-abdominal-thrust" picture, the glottis opening and shutting during the operation to allow of forceful, explosive emission of air. During the periods when the glottis is shut, the pressure in the thorax rises and blood is driven out to the muscles, in the manner which has been described above, producing the effort-picture seen in public speakers, which is essentially the effort-picture we have been describing and discussing. In Fig. 22 the inward thrust of the abdominal muscles is well shown. It occurs at the same moment at

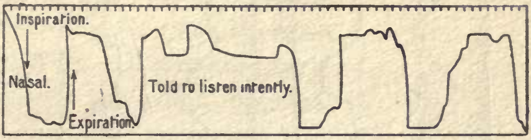


FIG. 18.

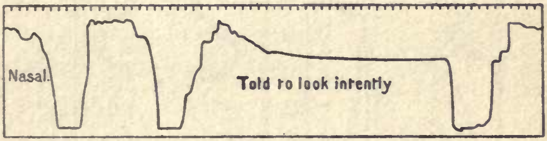


FIG. 19.

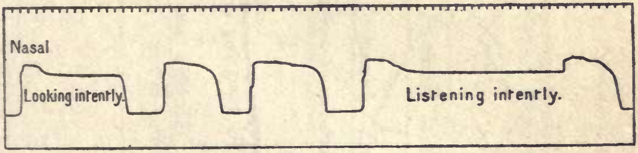


FIG. 20.

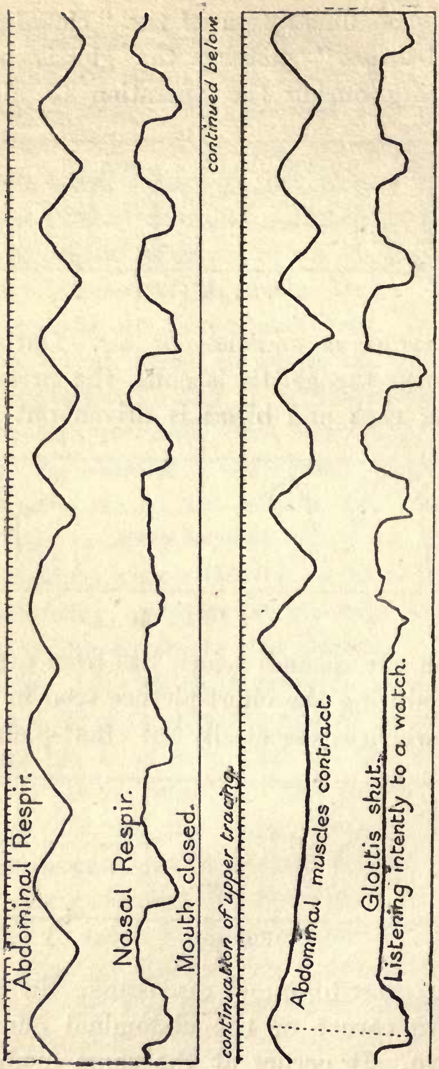

Abdominal Respir.
Nasal Respir.
Mouth closed.
continued below.
continuation of upper tracing.
Abdominal muscles contract.
Glottis shut.
Listening intently to a watch.

FIG. 21.

which the nasal respiration is inhibited and the subject begins to speak.

All the more strenuous Efforts of Life reveal the same Mechanism at Work.—It is unnecessary to labour the point, because any one may carry out the observations for himself. It is a matter of common and universal knowledge that the "breath is held" during strong efforts, and that the abdomen draws inwards until the breath is released with a puff or grunt. Every runner holds his breath in the last lap of a race, when the supreme effort is required. The "heave of the shoulders," so often described, is invariably accompanied by a holding of the breath and an action of the abdominal muscles. Coughing is evidently accomplished by the same mechanism, and likewise also laughter and crying.

The mechanism is, in these lesser activities, superimposed

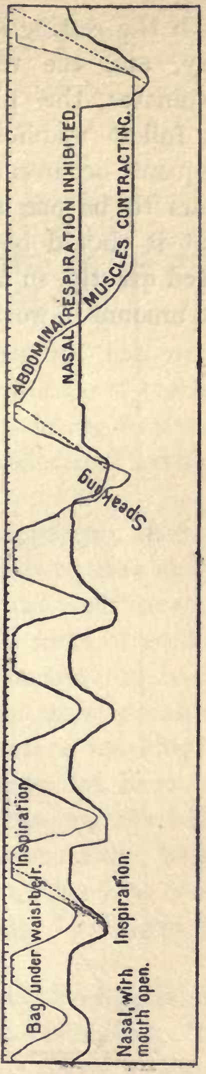


FIG. 22.

upon the resting state only to the degree necessary, and the resting state in other respects dominates the picture. The reaction state in its fullest manifestations is necessarily not of frequent occurrence in ordinary life, as it appears to be one of the conditions of healthy life that it should be controlled and restrained and fitted exactly, so far as its extent is concerned, to the amount of work requiring to be done.

CHAPTER V

THE ANATOMY OF THE ABDOMEN IN RELATION TO THE REACTION STATE

THE foregoing observations led the writer, who had enjoyed the advantage of teaching anatomy in the dissecting-room during five years, to study the contents of the abdominal cavity in relation to the reaction state.

The first thing that became apparent, after this investigation was begun, was that—to use a method of speech—the reaction state was well known inside the abdominal cavity. All sorts of evidence pointed to the fact that the various organs had played their parts in this state on many occasions.

There were, for example, surface markings on the organs which would certainly not have been there had the organs spent life hanging like berries from a tree, but which would, as certainly, be produced if they were all thrust together into a solid mass and jammed up against the diaphragm by a powerful muscular thrust.

Again, the shape of the cavity itself was sufficiently remarkable to arrest attention. That shape, as viewed from the side, is shown in Fig. 23.

It will be noticed that the rectus muscle is brought at the point L very near to the lumbar curve of the spine, and that in this way the abdomen is divided roughly into an upper chamber and a lower, the pelvis. At the top of the upper chamber is the diaphragm.

The next point of interest is the transverse section of the abdomen at the region of the costal margins, as shown in Fig. 24. This cavity, it will be seen, is divided by the vertebral column into two lateral wings in such a manner that the "piston" of the "syringe"—the massed abdominal viscera —must, when forced into it, tend to be divided into two laterally placed pistons, each one operating directly upon the lung situated above it and fitted over it (Fig. 25). One of the effects of this must obviously be to bring the structures composing each "piston," *i.e.*, the viscera upon

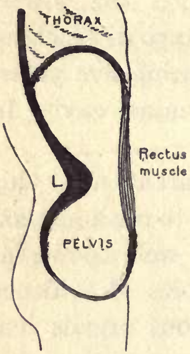


FIG. 23.

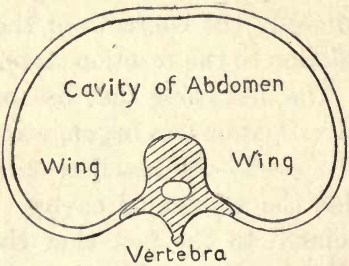


FIG. 24.

each side of the abdomen, into closer apposition than could occur if the division of the cavity by means of the vertebral column had not taken place. The kidney, on either side, for example, must be thrust up directly and forcibly against the solid organ above it; on the right side the liver, on the left, the spleen. But, as the suprarenal capsules surmount the kidneys, these structures must be forcibly squeezed between the kidneys, liver, and spleen during each reaction state. In point of fact, on both liver and spleen pressure marks made by the suprarenal capsules can be observed on the cadaver. Schäfer describes that on the liver as—"a triangular impression produced by a portion of the right suprarenal gland which projects upwards from the superior extremity of the right kidney between the diaphragm and liver." Quain describes the markings on the left suprarenal capsule as—"the anterior surface (of the capsule) lies in contact with the upper end of the renal surface of the spleen and stomach. The

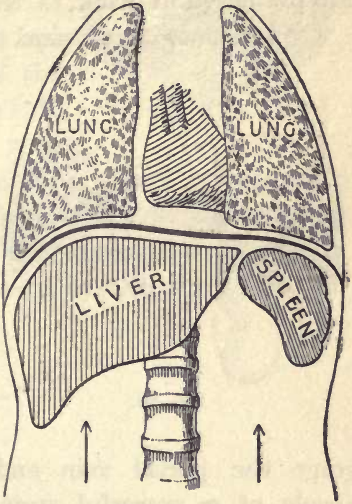


FIG. 25.

posterior surface is divided into two parts . . . the area median to the ridge looking in and back and resting on the left crus of the diaphragm."

This matter is of importance, as will be evident at a later period.

In Fig. 26 the relationship between liver, spleen, and kidneys is shown. The suprarenal capsules are presented in black.

The Intestines are squeezed and the Mesenteric Blood Lake is emptied into the Portal System.—The effect of the emptying of the mesenteric blood lake, which necessarily occurs when the intestines are thrust up under the liver and squeezed there, is, of course, to engorge the portal vein and convert it into the nozzle of a powerful syringe whereby the liver will be flushed with blood and the great veins rendered hyper-congested. It should be noted here that this portal blood lies *inside the barrel of the syringe*, and so is subjected to the full pressure of the abdominal wall just as the pulmonary blood is.

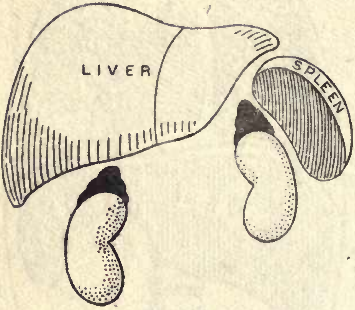


FIG. 26.

The direct squeeze upon the stomach, a part of the wall of which is in contact with the abdominal wall,

renders vomiting probable in sharp Reaction States.—The stomach as a hollow organ must be heavily squeezed in sharp reaction states. As the pylorus lies for the most part on the vertebral column, against which it will be directly squeezed by the abdominal wall, and as the thrust of the abdominal wall is upwards, the contents of the stomach, if the organ is full, will tend to be ejected with some violence. This, in point of fact, is frequently observed to occur in sharp states of reaction. In Fig. 27 it is seen how the stomach is practically closed at the pyloric end by the thrust of the abdominal wall.

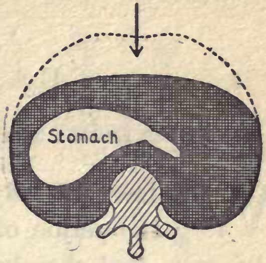


FIG. 27.

CHAPTER VI

THE EMPTYING OF THE ABDOMINAL BLOOD CISTERN

WE have already seen that, on account of the action of the oblique muscles of the abdomen, that chamber becomes, as it were, a continuation of the thorax, both thorax and abdomen forming the barrel of a syringe of which the recti muscles are the motive or emptying force.

It is exceedingly important that this truth should be clearly mastered because unless it is understood a totally wrong conception of the reaction state will arise. If it is understood it will at once be evident that the same pressure which is exercised on the thorax is exercised also on the abdomen the moment the recti muscles begin to thrust, and that every part of the abdomen affected by these muscles feels the thrust. The blood in the mesenteric or abdominal blood cistern is, therefore, under as strong compulsion to " get out " of the barrel of the syringe as is the blood in the lungs.

In other words, there *is a through blood wave beginning at the mesenteric lake, passing up the portal system, through the liver to the inferior vena cava, and* *thence through the right heart to the lungs, and so with force to the left heart and the arteries.*

The meaning of this is that not only the pulmonary blood cistern is emptied into the arterial tree, the abdominal blood system — portal system — is also so emptied at the same moment, and the two blood masses, mingling in their course, are hurried together to the arteries and so to the muscles and cerebral cavity. *The portal blood flushes out the liver at high pressure on its way to the muscles.*

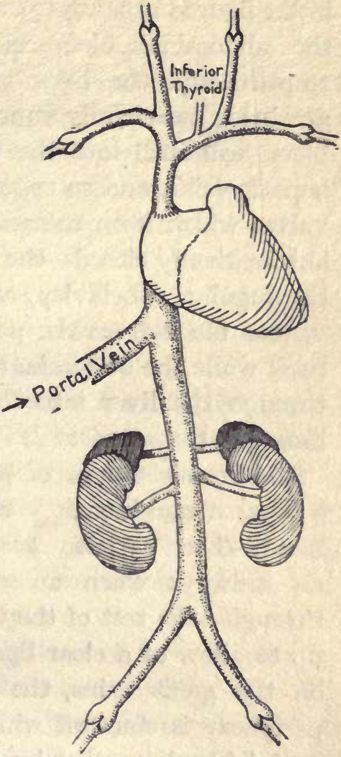


FIG. 28.

The Arrangement of Valves on the Great Veins forms a Cistern known as Keith's Cistern.—Some time ago Professor Keith described the manner in which the valves of the great veins produce a kind of venous cistern within the trunk of the body. This is illustrated in Fig. 28. It will be seen from the illustration that opening into this cistern and unvalved are the hepatic veins (marked "Portal Vein" to show the continuity from that structure), the renal and suprarenal veins (the latter figured separately). It is evident that when the abdominal blood cistern is being forcibly emptied and the liver and suprarenal capsules are being powerfully squeezed, a large volume of blood will rush into the venous cistern from the hepatic veins, and so engorge that cistern that the valves which form the walls of the cistern will be automatically closed—the valves, that is to say, on the jugular, subclavian, and femoral veins. Thus venous blood from the periphery will be dammed back while the abdominal blood cistern is emptied through the liver into the heart and so, by the lungs, to the arteries.

The Venous Cistern of Keith is a means whereby a clear "run-through" is afforded to the Portal Blood.—The position resembles that met with on railways when an express train is passing through—the rest of the traffic is temporarily held up to allow of a clear line. Thanks to the valves on the great veins, the venous blood from the periphery is shut off while the main "surge" of portal blood is going by, issuing, that is, from the liver by the hepatic veins. Thus a mechanism exists whereby the "scourings of the liver," be they glycogen or other substance, are conveyed to the arteries and so to the muscles with the utmost possible rapidity and with the least possible dilution.

What applies to the Portal Blood applies also to the Suprarenal Blood, and in a lesser degree to the Blood from the Pancreas. — We saw that the suprarenal capsules were forcibly squeezed between the kidneys and the liver and spleen. This must tend to drive out their venous contents, and the venous contents entering the venous chamber of Keith will pass with the portal blood to the arteries—in other words, suprarenal blood will be afforded " a seat in the express train." Owing to its deeply placed position the pancreas will tend to feel this pressure strain less. Yet in long continued reaction states this organ too will give of its content to the portal blood.

By this means Venous Return from the Periphery—especially the Muscles—will be as good as stopped.—When all the valves of the venous chamber or cistern of Keith are shut venous return from the periphery will be stopped. The effect of this will necessarily be to engorge the muscles of the body with blood and greatly to raise blood pressure throughout the whole system of veins, capillaries, and arteries supplying these muscles (Fig. 29). The muscles, in fact, will lie in a high-pressure blood bath which, on its arterial side, is being enriched by the " scourings of the liver " and the contents of the suprarenal and other bodies brought by the " express train " from the venous chamber of Keith.

Another important Vein opens into the Venous

Cistern of Keith—the Inferior Thyroid Vein.—This vein enters the innominate vein, and so opens directly into the chamber. It is not valved.

We have already seen that, at the moment of reaction, the head is thrown back and the sterno-mastoid muscles go into powerful contraction. Powerful contraction of the sterno-mastoid muscles with the head thrown back must strongly compress the thyroid gland against the vertebral column (Figs. 30 and 31). This pressure must necessarily tend to drive out the contents of the gland, and the only means of exit available will be the thyroid veins. The inferior thyroid veins with their plexus will thus afford a ready means of exit into the venous chamber of Keith. We may take it, then, that along with the "scourings of the liver" and the suprarenal vein content, thyroid vein content will be driven onwards into the muscles during the reaction state.

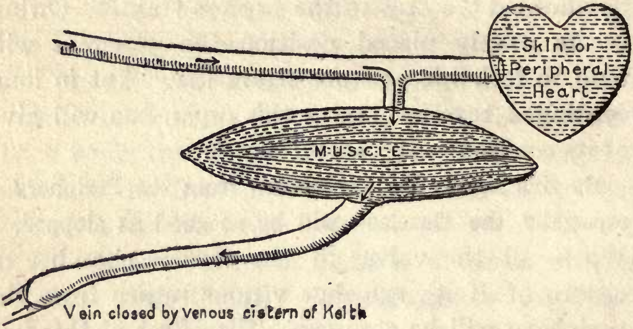


FIG. 29.

There is another substance, in addition to Air and Blood, which can conceivably be driven out of the Abdomino-thoracic Chamber—the Syringe Barrel—by the action of the Abdominal Wall Muscles—Water.— The kidneys constitute a filter by which water can be removed from the blood. So that just as there is a tendency to force air out of the lungs, and blood out of the blood cisterns, and its contents out of the stomach, so must there be a tendency to force water out of the blood by means of the only available channel, the kidney filter.

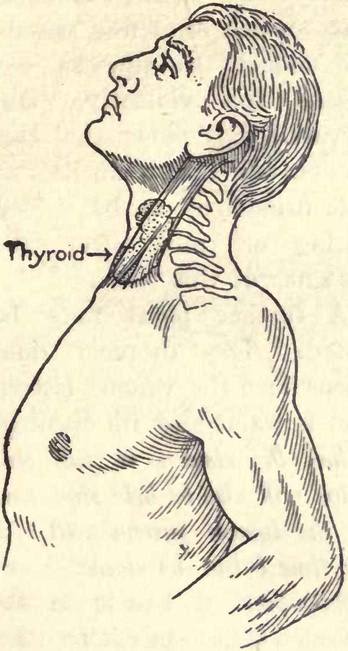


FIG. 30.

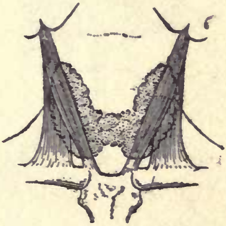


FIG. 31.

The kidney veins, as we saw, open unvalved into the venous cistern of Keith, so that as the pressure in that chamber rises there will be an increasing tendency for fluid to escape out of the "barrel of the syringe" by any other available route. In Fig. 32 the other available route is shown diagrammatically. We might therefore expect unusual activity of the renal filter during reaction states which were not powerful enough to squeeze the organs too violently. Observation has shown that this occurs, and that quantities of pale urine tend to be voided during or just after the presence of this state.

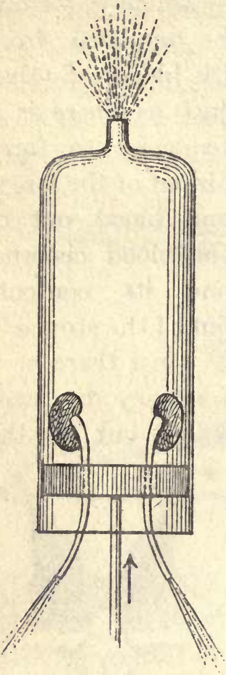


FIG. 32.

A further point may be noted. The thoracic duct opens into the venous cistern and is valved at its opening. *When the cistern is full this valve will also be held shut, and so the lymph system will, for the time being, be closed.*

Summary of Events in the Reaction State.—In the reaction state, therefore, the blood cisterns of the lungs, abdomen, and periphery are emptied. The blood is driven out into the muscles and brain. The mechanism by which this is accomplished is of such a nature that the contents of the liver, the suprarenal glands and the thyroid gland, also the spleen, are specially collected in the great venous cistern described by Keith, and are specially hurried on through the thorax into the arteries, and so to the muscles, etc. In prolonged cases pancreatic contents will also be present. Meanwhile the same mechanism tends to force water out of the kidney. The essentials of the mechanism are a rigid condition of muscles producing a syringe-barrel-like trunk, a closed or intermittently opening glottis to prevent the outlet of air, a " piston " in the shape of the abdominal viscera, and a powerful drive, supplied by the muscles of the abdominal wall. Respiration in the sense of gaseous exchange is stopped or greatly reduced during this reaction state. The air in the lungs serves not as a means of oxygenation but as a pneumatic buffer, as has been described above.

CHAPTER VII

REST BREATHING

OBSERVATION has shown that the instant the reaction state comes to an end another state of the body of an utterly different type succeeds it.

The subject at once relaxes all his muscles. The head comes forward and even tends to droop. The shoulders fall away, and the scapulæ move outwards, lying flat upon the ribs. The thorax, released from the elevating muscles, sinks down, the costal margins again approximate to one another, and the angle between them becomes more acute. The chest assumes a more barrel-shaped and a less funnel-shaped appearance. The abdominal muscles lose their tone and bulge out flabbily. At the same time the whole body loses its stiffened appearance and tends to stoop forwards, the knees appear to bend a little and often a pink flush overspreads the skin.

At the same moment the subject as a rule inspires deeply and long, producing the effect which is described as a " sigh of relief." This sigh may be repeated for a time, but the tendency sooner or later is for the inspiratory periods to become shorter and the expiratory periods to become longer. The thorax plays little part in this inspiration; the abdominal muscles, now obviously flabby, move out in front of the descending viscera. It is obvious to any observer that the inspiration is caused chiefly or wholly by the descent of the diaphragm.

(The gradual lengthening of expiration after exercise is shown in **Fig. 33.**)

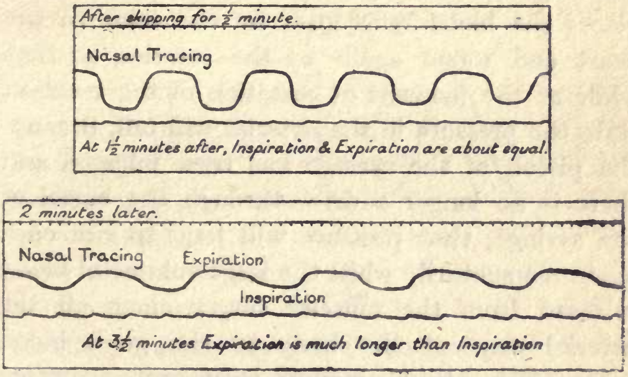


FIG. 33.

The Effect of Rest Breathing is exactly opposite to that of Reaction Breathing.—The first effect of rest breathing is to draw back the piston of the syringe, and so to reverse the expulsive process and convert it into a suction process—though it is not sought to labour this latter point. As the diaphragm descends the intrathoracic pressure falls. Air rushes in to fill up the space, and so also does blood. The valves of the venous cistern of Keith are opened. The mesenteric vessels are no longer compressed, and so the huge abdominal blood cistern is able to receive blood. Likewise also the pulmonic blood cistern receives blood again. The skin flushes and is filled with blood.

Thus at once the muscles and brain are drained of blood, which in great volume returns to its lakes or cisterns in the mesenteric vessels and capillaries, the lungs and the skin. In order to reach these places the blood must pass by the veins to the heart and round again by the arteries, so that while at the moment of cessation of the reaction state the pressure in the arteries will fall, because the piston of the syringe has been released and there is no longer a drive through the barrel of the syringe, that pressure will tend to rise once again momentarily while the large volume of blood released from the muscles surges along all the arterial ways of the body to the great blood lakes. The blood pressure curve will therefore show, as in Fig. 34, when the pressure is taken on a systemic artery like the radial. This view would seem to be in consonance with the recent work of Rapport reported from Mount Vernon Hospital, Hampstead.

Rest Breathing consists of Expiration and Inspiration.—Rest breathing or normal breathing consists of expiration and inspiration. These have been observed and described so often that it is unnecessary to dwell upon them. Enough that in inspiration the abdominal wall bulges out during the easy descent of the diaphragm, and in expiration moves inwards again in virtue of its elastic recoil.

The Abdominal Wall is inhibited in Rest Breathing.—This can be determined by observation. The muscles of the wall are absolutely soft and flabby, they "float up" on the abdominal contents, and on being touched are found to be soft and toneless. Not a fibre of them appears to act. At this time, too, the glottis is never closed.

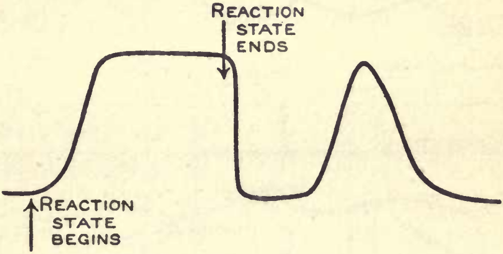


FIG. 34.

In the Rest State the Inspiratory Phase of Respiration is the Active Phase.—We saw that in the reaction state the expiratory phase was the active phase of respiration. The exact opposite holds good for the rest state. In this state expiration is a period of absolute quiescence "with not a muscle moving"; on the other hand, in inspiration the diaphragm is contracting, and consequently a certain amount of additional work is being performed.

The Descent of the Diaphragm sucks blood into the Thorax, and so, momentarily, and to a slight degree, raises the arterial pressure by increasing the volume of blood passing to the arteries and also quickens the pulse rate.—The descent of the diaphragm is like the pulling back of the piston of a syringe. Blood and air are sucked into the thorax, the former by the great veins. This necessarily entails an increased output of blood by the arteries, and the effect of it in certain cases may be seen in a slight increase of pulse rate during normal, quiet inspiration (so-called sinus arrhythmia); see Fig. 35. The arterial blood pressure is also raised, as may be observed on any blood pressure tracing taken by a cannula in the artery—the so-called inspiratory wave of arterial pressure.

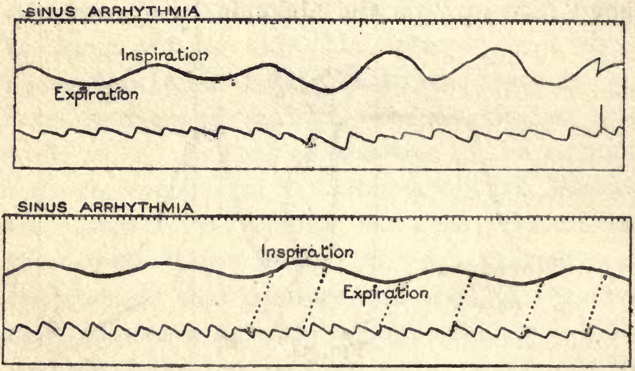


FIG. 35.

The descent of the diaphragm will also tend to give the suprarenal capsules, both of which impinge upon the diaphragm, a slight squeeze, so that during normal inspiration these organs are probably caused to deliver some of their content into the venous blood. In any case the venous blood from the whole body tends to be siphoned more rapidly into the thorax at this moment.

In reaction breathing, then, to recapitulate, expiration is the forceful phase of respiration, while

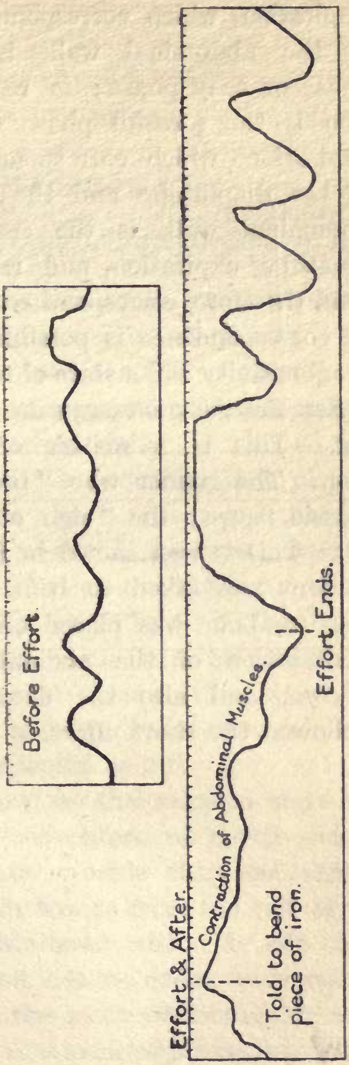


FIG. 36.

inspiration, which corresponds to the relaxation of the abdominal wall, is the slackening-off phase or easy phase; in rest breathing inspiration is the forceful phase of respiration, while expiration, which corresponds to the slackening of the diaphragm and the elastic recoil of the abdominal wall, is the easy phase. Reaction breathing expiration, and rest breathing inspiration, therefore, correspond to one another in so far as correspondence is possible between a state of great activity and a state of relaxation and rest.

Rest Breathing is exaggerated after Reaction Breathing.—This is a matter of universal observation. The runner who "breathes himself" is a classic figure; the "sigh of relief" is known to all. This is well shown in Fig. 36, in which the patient was asked to bend a piece of iron. A bag tambour was placed on the abdomen. The contraction of the abdominal muscles is well shown, and also the deeper breathing which followed the short effort.

CHAPTER VIII

EVENTS IN THE CRANIUM DURING REACTION

WE have so far made little mention of the effect of the reaction state upon the brain except to point out that every attempt at close thinking or clear perception is accompanied by evidence of the reaction state.

In coming now to the consideration of this question it is necessary to bear in mind that the skull is a closed box, and that the vascular elements are so arranged that the veins of the inside of the cranium pulsate with each beat of the heart. Thus, were the venous return from the cranium to be retarded, there must come a time when the arterial blood could no longer enter the brain box at all—the brain box would be full.

We have seen that in the reaction state the presence of the venous cistern of Keith and its closure in that state provide that the venous return from the brain box as from the rest of the periphery will be interfered with. At the same moment arterial blood is being driven with greatly augmented force up the great arteries of the neck into the brain box. It is manifest, then, that unless room is found for this incoming blood it cannot come in at all after a certain point. The incoming arterial blood will therefore at first, in virtue of its high pressure, sweep out before it the cranial content in the shape of the cerebro-spinal fluid. This is illustrated diagrammatically in **Fig. 37**, where C.S.F. = the cerebro-spinal fluid. Naturally as the venous pressure rises the sweeping-out process will decline—though, of course, the point at which no arterial blood can enter the cranium is not reached in actual life—it is merely a hypothetical point.

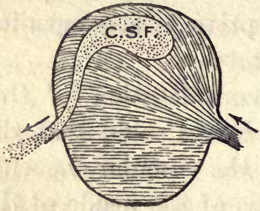


FIG. 37.

Consequently, it is evident that cerebro-spinal fluid will be passed out of the cranium along with the venous blood during these periods of high pressure. In this connection it is interesting to note that Dixon and Halliburton found (*Journal of Physiology*, vol. i. No. 3, p. 199 *et seq.*) that "substances introduced into the cerebro-spinal fluid which can be readily traced do not find an exit by the lymph channels but appear rapidly in the blood. . . . It (the diffusion process) probably occurs into the venous sinuses by the microscopic arachnoid villi, but Mott's view that the transference partly occurs through the thin walls of the blood vessels within the central nervous system is not excluded by our experiments, seeing that contact of these vessels with the cerebro-spinal fluid is maintained throughout their extent by the perivascular spaces which are continuous with the subarachnoid cavity."

The increased intracranial pressure during the reaction state can be studied easily in the fontanelles of a baby's skull when a baby is crying. The writer has also had opportunities of studying it in the case of a friend who lost a portion of cranial bone by a shell wound. Each time he coughed the soft parts bulged out through the wound. The act of coughing, as has been shown, is a picture of the reaction state.

During the Period of Intracranial Pressure in the Reaction State the Pituitary Body in the Sella Turcica is bound after a time to be subjected to strong pressure along with the other Cranial Contents.— The pituitary body is so situated that increase of intracranial blood pressure must squeeze it. It may be said to lie between the two cavernous sinuses, or rather in the middle of a large venous lake (Fig. 38). Its contents will, therefore, tend to pass up the infundibulum and to be carried away in the cerebro-spinal fluid during the general emptying of the cranial cavity before the high pressure sweep-out of the arterial blood; or, alternatively, its contents may be ejected into the blood stream and so carried away. In any case, the gland, like the contents of the cerebrum, will be subjected to pressure, and this principally in the later and more extreme states and stages of reaction.

It has been stated that when the circulation of blood in the Cranium becomes unduly slow, owing to restriction of Venous Return, the Respiratory Centre is stimulated and inspiration deepened, thus relieving the pressure.—This statement clearly does not recognize two separate types of respiration such as our observations have led us to believe exist. On the other hand, it does suggest a mechanism whereby the reaction state can be modified and "diluted," so to speak, and the rest state ushered in to replace it. We do not think, as we shall hope to show, that this excitement of the respiratory centre is by any means the whole mechanism whereby the reaction state is modified and controlled, but it is clearly a part of that mechanism.

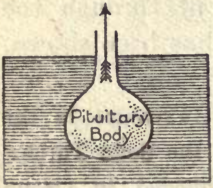


FIG. 38.

The Rest State is excited by the Reaction State, and acts therefore as a kind of Governor of that State.—Apparently, as we have seen, reaction is always followed by an exaggerated "rest" picture, *i.e.*, by fatigue. It is also, it would seem, always modified by the rest picture in a healthy man, so that, except at extreme moments, it is never presented in a pure state. To excite the reaction state is therefore also to excite the rest state as soon as a certain level of violence has been reached. Action and reaction, indeed, seem to be equal and opposite. In many conditions of ill-health the rest state follows the reaction state in so active a manner that a kind of collapse follows effort or excitement, and careful study has been made of these cases.

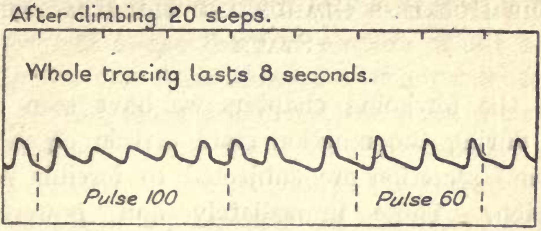


FIG. 39.

In Fig. 39 the pulse of one of these patients is shown, and it will be observed that the slowing which occurs is sudden and violent. Some patients have been observed to faint during this period of excessive exhibition of the rest state, or in other language of " vagus pull."

CHAPTER IX

The Ductless Glands and the Reaction State

IN the foregoing chapters we have seen that during the reaction state certain organs of internal secretion are subjected to forcible compression. Those immediately and powerfully compressed are :—

The Suprarenal Capsules, by the thrust of the abdominal muscles.

The Thyroid, by the contraction of the sterno-mastoids.

The Pituitary, by the gradually rising pressure in the cranial venous sinuses.

The Pancreas, by the gradually increasing pressure in the abdomen as the result of the continued upward thrust of the abdominal muscles.

The Ductless Glands are grouped according to the immediate or later Exercise of Force tending to compress them.—We are therefore entitled to group the ductless glands in two classes : those which are subjected in the reaction state to immediate, strong compression, and those which are less likely to be compressed at first, but are bound to be compressed strongly if the reaction state is continued and carried on for a prolonged period. Into the first group go, as we have seen, the thyroid and suprarenals; into the second go the pancreas and pituitary.

The Pancreas is so placed in the Abdominal Cavity that it will escape the first pressure of the upward thrust of the Recti Muscles. — The pancreas lies deeply and extra-peritoneally in such a position that pressure from the squeeze of the abdominal wall can only reach it through the bowels heaped up on top of it; this pressure will therefore reach it through an "air buffer" ***i.e.***, the air in the bowels. It is therefore evident that while the organ must be compressed along with the other viscera by the generally increased intra-abdominal tension, it will be less subject than the other viscera to a direct squeeze; it will receive a direct squeeze later than the other viscera, and only when the reaction state has lasted so long that the extreme limits of it are being reached.

The Pituitary Body is so placed in the Cranium that it will escape the first effects of the rising Blood Pressure in the Arteries. — The position of the pituitary body under its own roof, so to speak, ensures that until the great venous sinuses of the cranium are filled full it will not be subjected to direct squeeze. Like the pancreas, it will, therefore, not be forced to give up its contents until the later stages of reaction have filled all the venous sinuses full and so caused the venous pressure in the cavernous sinuses to exercise direct compression upon the pituitary body.

We can, therefore, say with some certainty that—

During the reaction state, adrenalin and thyroid will be present in the blood from the outset.

In the more extreme manifestations of the state, pituitary extract and the internal secretion of the pancreas will tend to make their appearance, and will continue to present themselves from this time forward in increasing amounts

CHAPTER X

The Effects of Adrenalin

WE must therefore make ourselves familiar with the action of adrenalin in the first instance, so as to see whether or not its presence in the blood during reaction states will militate against or for the efficacy of these states.

The writer enjoyed a large number of opportunities of administering adrenalin intravenously both to healthy and to sick subjects (work accomplished with Dr. F. R. Fraser and other workers), and the following may be taken as an effect observed after a dose of 0·01 mgrm. of 1 to 1000 solution.

The patient blanched perceptibly, and the breathing became in many instances a little "irregular." The glottis tended to close, as was revealed by the staccato character of the expiration following the dose ; the muscles twitched and the patient seemed restless. The pulse was quickened, about 28 beats a minute for a short period, the blood-pressure raised from 21 to 25 mm. of mercury, the vessels in the neck filled perceptibly.

It will be seen that this effect, so far as it goes, bears a striking resembance to the reaction state which we have described, and affords grounds for the idea that adrenalin, being squeezed into the blood during this stage, will help to sustain the reaction picture.

The Detailed Physiological Work which has been carried out on Adrenalin goes to show that this Substance plays an important Part in sustaining the Reaction State.—An enormous mass of physiological work has been carried out in order to elucidate the effects of adrenalin on the various structures of the body. The conclusion of the whole matter is summarized by Langley, who has declared that the effect of adrenalin is always the same as the effect of stimulating the true sympathetic (as opposed to the vago-sacral sympathetic). Wherever fibres of the true sympathetic go there adrenalin acts, whether the tissue be in its proper place in the body or be removed from the body. If stimulation of the true sympathetic produces action of the tissue—*e.g.*, vaso-constriction, then adrenalin produces the same action; if stimulation of the true sympathetic produces inhibition, *e.g.*, dilation of the bronchioles, then adrenalin produces inhibition. Further, on tissues which have no nervous supply from the true sympathetic adrenalin produces no action of any kind. The action of adrenalin is therefore a peripheral action upon nerve endings (upon the "neuro-muscular junctions," to be exact). The drug is inert without these neuro-muscular junctions of the true sympathetic system; the neuro-muscular junctions of the true sympathetic system appear to be inert without the drug. So we arrive at a kind of equation:—

$$a+b=\mathrm{C}\ (\text{Reaction});$$

where a is the neuro-muscular junction of the true sympathetic system and b is adrenalin. Without "a" there can be no reaction and without "b" there can be no reaction. Both are necessary to the production of "C."

The Emptying of Blood from the Blood Lakes or Cisterns of the Body during the Reaction State is powerfully assisted by the action of Adrenalin.—We have seen that the first blood lake to feel the effect of the upward thrust of the abdominal wall muscles is the mesenteric blood lake, that huge reservoir of blood which, as the famous experiment of Salathe on the hutch rabbit showed, can accommodate almost all the blood in the body.

We have already considered the emptying of this blood lake as a purely pressor phenomenon, *i.e.*, as the result of a direct physical squeeze. We may now discuss the assistance rendered to this physical means by the neuro-muscular action of adrenalin.

We note, in the first place, that the vessels of the mesenteric blood lake, the arterioles especially, are supplied with constrictor nerves by the true sympathetic ("a"); and we note, in the second place (Schäfer, *Endocrine Organs*, p. 59), that "the arteries which are most affected" by adrenalin "are those of the splanchnic area." So that it is evident that during an outpouring of adrenalin ("*b*") these arteries of the splanchnic area, or abdominal blood lake, will be powerfully contracted and the blood actively dislodged into the portal vein.

Thus in this respect the action of adrenalin corresponds to and powerfully augments the action of the abdominal muscles in their upward thrust in the reaction state.

Further, it has been shown many times, and is even accepted as a test, that adrenalin paralyses the muscles of the bowel wall (which are likewise inhibited by the true sympathetic). The effect of this is to stop peristalsis and render the bowel inactive—*i.e.*, to render it a most suitable structure for being compressed by the upward thrust of the abdominal wall.

Adrenalin increases the Heart Rate.—We saw that in the reaction state the rate of the heart is always increased. It is a universally acknowledged observation that the effect of adrenalin is to increase the rate of the heart, and in all the intravenous work which the writer took part in the rate of the heart was increased after administration of the drug.

The effect is produced by action upon the neuromuscular endings of the true sympathetic. But we have seen that the thrust of the abdominal muscles and the great blood wave rushing through the thorax will also tend to accelerate the heart rate by filling the organ more forcibly in the diastole and by assisting the systole.

The sympathetic nerves to the heart have been called by Luciani and others "the nerves of systole," because during their action diastole is shortened and limited in extent, whereas the numbers of systoles are increased.

Adrenalin alters the Respiratory Phenomena.—This was well seen in some of the cases observed by the writer. There was a certain diminution in the depth of the respiration, and sometimes pauses occurred which were due to the closure of the glottis. The pressure effects of the abdominal musculature were well seen at these times, the muscles standing out and the patient appearing "uneasy." Schäfer records the same findings, saying (*Endocrine Organs*, p. 59): "There is some diminution in the depths of the respirations and occasionally a temporary cessation of breathing, especially in the rabbit. But this disappears long before the effect on the blood vessels has ceased to show itself. The effect on the blood vessels lasts a few minutes."

The effect of adrenalin by injection is exceedingly transitory, as might be expected, for the reaction state is limited very strictly in duration to the "need of the moment."

This is shown in Fig. 40.

Adrenalin dilates the Bronchioles.—Upon this observation has been founded the treatment of asthma by adrenalin injections.

We may recall that observations showed that the reaction state was ushered in by a short "gasp" or shallow, sudden intake of air, and that it was upon the "buffer of air" thus procured that the thrust of the abdominal wall acted. This gasp will necessarily be most effective the more widely dilated are the air channels, and that for the simple reason that if the bronchi were not widely dilated the sudden, forcible opening of the chest would create a negative pressure in the thorax (because the air was rushing in at a slower rate than room for it was being made). This would at once cause blood to be sucked into the thorax, as occurs, indeed, in rest breathing, and so would defeat the mechanism of the reaction state. We see here, then, a further evidence of the assistance given by adrenalin to the perfecting of the reaction state.

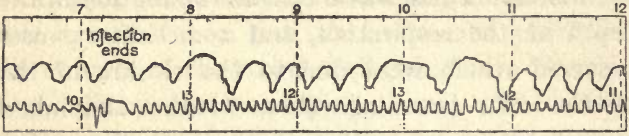


FIG. 40.—An Injection of Adrenalin.

Adrenalin empties the Peripheral Blood Lake.—The blanching observed after an injection of adrenalin is evident for all to see. Further, it is a universal observation with this drug that the arterioles of the periphery are caused to constrict by it. More recently it has been suggested that the capillaries of the skin are under nervous control, and can be constricted by adrenalin (a suggestion made earlier in the day by Roy), and several observers have described the presence of nerves to the capillaries.

Further, Gunn and Chevasse showed that adrenalin constricts the *superficial* veins throughout the body.

Our own observations have shown that in reaction states the superficial veins tend to be greatly constricted, and this is also seen after intravenous administration of adrenalin.

Here, then, we find the emptying of another great blood lake by adrenalin—the peripheral blood lake or skin. We have already suggested that a similar emptying of the peripheral blood lake occurs in the reaction state. The action of adrenalin is thus once again found to augment and assist the full development of the reaction state.

The Action of Adrenalin on the Pulmonary Blood Lake is less than its action on the Abdominal Blood Lake and the Peripheral Blood Lake.—We saw that during the reaction state a large blood wave was thrust through the portal circulation, through the venous chamber of Keith, through the right heart, through the lungs, and so to the left heart and the arteries by the contraction of the recti muscles.

This implies an eventual emptying of the lungs of blood, as we have seen. But it also implies a free passage-way for the blood wave through the lungs, for if such passage-way did not exist the blood wave would be dammed up and the thrust of the abdominal wall would either burst the lung vessels or the right heart, or would fail altogether to effect its purpose. This is self-evident.

Now it has for long been a disputed point whether or not adrenalin exercised any effect on the pulmonary vessels, especially the arterioles of the lungs, and the bulk of the evidence is against any strong action on these tissues by adrenalin. Schäfer states that the arteries of the pulmonary system are "only very slightly if at all affected." This view, however, has been disputed by Gaskell, whose conception of the lungs as an appendage of the alimentary canal led him to suppose that the effect of adrenalin on the mesenteric vessels would be repeated on the lung vessels. He writes (*The Involuntary Nervous System*, p. 48):—

"Plumier and Wiggins found a distinct diminution in the rate of flow through the lungs when fluid containing adrenalin was sent through. Fühner and Starling also obtained a distinct rise of pressure in the pulmonary artery by adding adrenalin to the perfused blood in the heart-lung preparation. Tribe also found that if the pressure in the pulmonary artery was not above the normal, adrenalin always caused a marked diminution of flow through the lung."

It is probable that this last observation supplies the key to the puzzle. There would seem to be a tonic effect of adrenalin on the smaller pulmonary vessels. If the pressure in the pulmonary artery is high—as it certainly is in the reaction state—this tonic effect has no influence on the through-and-through blood flow. Thus the adrenalin effect, while not interfering with the passage of blood, gives a tonic stimulus to the vessels, and so aids the process of passing the blood wave onwards by reducing the size of the pulmonary lake and converting the lake into a series of rivers by canalizing it, so to speak. This is illustrated diagrammatically in Fig. 41.

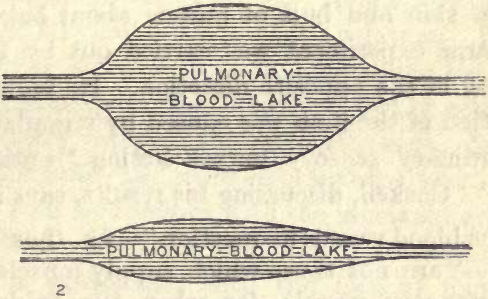


FIG. 41.

Thus, in respect of its so-called "atypical" action on the pulmonary vessels, adrenalin materially assists the effectiveness of the reaction state and in no wise hinders it.

Adrenalin has no constricting action on the Vessels to Muscles, but has the contrary effect.—This observation has recently been confirmed by workers in America in the following manner. The leg of an animal was placed in the plethysmograph and adrenalin administered. A shrinkage of the limb volume resulted. The thick skin was now removed and the experiment repeated. The increase in the limb volume resulted (a point was chosen where bulk of skin and bulk of muscle about balanced). The same experiment was carried out by Bayliss working in the opposite direction. He found that dilatation of the limb was caused by stimulation of the ordinary sensory nerves acting "antidromically." Gaskell, discussing his results, says :—

"The blood vessels in question"—*i.e.*, those which dilate—"are not those which supply muscles, but are cutaneous vessels, for when the ankles and feet only are put into the plethysmograph the effect is very large, and when the skin is removed the effect is gone."

Thus the agent adrenalin which constricts the skin vessels fills up the muscles with blood—as we noted must occur by purely physical means in the reaction state, while the stimulation of the sensory nerves antidromically which dilates the skin vessels in Bayliss's experiment has no effect in dilating the muscles.

A moment's consideration will lead to the question: Is this action of skin vessels as opposed to muscular vessels under adrenalin a mechanism whereby the blood dislodged from the peripheral blood lake or skin shall be thrust into the muscles? And, contrariwise, when the skin dilates are the muscles relieved of blood?

We saw in studying the reaction state that the blood dislodged from the great blood lakes must be driven into the muscles and brain because no other lodgment for it existed. Here is the manner in which adrenalin assists that operation. In this connection we must note that adrenalin has been shown again and again to have no sort of action on the coronary vessels or the cerebral vessels. Schäfer emphasizes this, and so do all other observers. This finding is in consonance with the increased activity of the heart observed after use of adrenalin, and is also in consonance with the idea of increased work which must fall upon the heart during reaction states.

Adrenalin raises the blood pressure in the Arteries and Deep Veins.—This again has been confirmed by countless experiments, and the writer has seen the result himself upon a large number of men who received intravenous injections. The mechanism is easily understood when it is remembered that adrenalin at the same moment is emptying the immense blood lakes—the abdominal blood lake and the peripheral blood lake. This huge added volume of blood, and the marked interference with venous return from the periphery occasioned by the rush of the blood from the abdominal blood lake through the portal system into the venous chamber of Keith (thus shutting the valves of the venous chamber and damming back the venous blood flow from the entire periphery), are together more than sufficient explanation of a sudden sharp rise of arterial pressure. Exactly the same thing is noted in the reaction state.

Adrenalin lowers Blood Pressure in the Peripheral Veins.—This follows from the fact that adrenalin closes the skin arterioles which pass their blood on to the skin veins. These veins become small and constricted in consequence.

Recent work by Captain Fairly Marris has shown that the peripheral venous blood pressure curve falls as the arterial pressure curve rises. This exactly confirms the view expounded above. *Care must be taken, however, to differentiate between the pressure in the superficial veins*—with which Captain Marris is dealing—*and pressure in the great, deep veins.* This latter, as we have seen, rises.

Adrenalin causes a flow of pale urine from the kidneys when the Pressor Effect on the kidneys is not too great. —This has been shown by various experimental workers.

We noticed that in reaction states a similar result took place, and we were able to show that the position of the renal filter in the " barrel " of the syringe while the abdominal muscles were exercising their upward thrust made such a leakage of water as certain as a leakage of blood or air from the barrel of the syringe was certain. If, however, the kidney is powerfully pressed upon, the leakage of water will be delayed until the pressure is slightly reduced, for the powerful pressure will shut the filter altogether.

It is interesting to note that, while intravenous injection of adrenalin diminishes the flow of urine for the moment (Schäfer), subcutaneous injection —a milder method—increases it forthwith.

This exactly corresponds to the picture seen in the reaction state.

Adrenalin is the Drug of the Reaction State.—The foregoing discussion entitles us to look upon adrenalin as the peculiar drug of the reaction state. We are therefore obliged to look also upon the true sympathetic nervous system as the nervous system of the reaction state, and so we get this equation :—

$$a \text{ (True Sympathetic)} + b \text{ (Adrenalin)} = \text{Reaction State.}$$

Further, it is interesting to note that we are in a position to say that this reaction state is produced, as it were, by a double means—by two separate means, working parallel to one another: by the purely physical mechanism of pressure, by the mechanism of the true sympathetic and adrenalin.

We may finally note that Sherrington has shown that stimulation of the splanchnic nerves (*i.e.*, true sympathetic fibres) throws the musculature of the abdominal wall into a state of *tonic contraction.*

CHAPTER XI

THE RELATIONS OF THE BLOOD LAKES TO THE HEART

CERTAIN very striking facts have emerged during the observations which have just been described, and these may now be set down.

In the first place, it has been made clear that during the reaction state certain definite groups of structures are activated. These are—

The Heart, which both by pressure of blood due to the thrust of the abdominal wall and by the action of adrenalin on the endings of the true sympathetic nerves is accelerated and brought into the condition which we may describe as "*condition of plus-systole*" (we may here recall the description of the sympathetic as the "nerve of systole").

The Mesenteric or Abdominal Blood Lake, which by pressure of the abdominal wall and by the action of adrenalin on its arterioles, etc., is emptied, and so also brought into a condition of plus-systole.

The Peripheral Blood Lake or Skin, which is emptied by adrenalin action, and so also passes into the condition of plus-systole, the arterioles, capillaries and superficial veins being all affected.

Fig. 42 shows this peripheral plus-systole occurring during inspiration in the rest state, which inspiration, as we observed, is the active phase of the rest state, and corresponds in some respects to expiration in the reaction state.

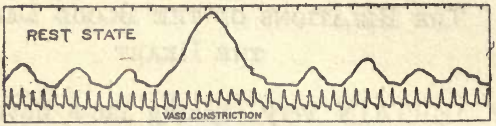


Fig. 42.

Fig. 43, which was taken from a man with malaria, temp. 100, in reaction to his fever, shows an exact reversal of the picture seen in Fig. 42. A very intense vaso-constriction follows the forced expiration. Then a short vaso-dilatation of three or four beats follows release of the tension in inspiration, and then the vaso-constriction begins again.

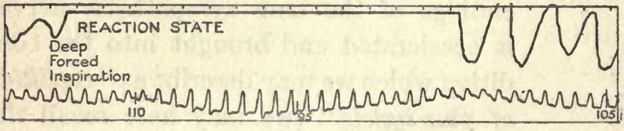


Fig. 43.

Fig. 44—the effect of inspiration on the patient with malaria when in full reaction—temp. 100, is shown. The effect, it will be seen, is a vaso-dilatation with the usual swing of the pendulum afterwards. This swing of the pendulum can be seen in both figures (43 and 44) and is very characteristic.

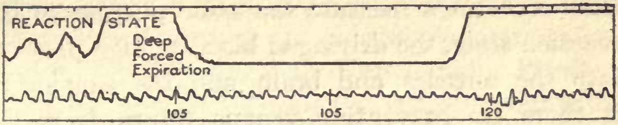


FIG. 44.

The Pulmonary Blood Lake, which is "toned" by adrenalin and emptied also by the thrust of the abdominal muscles. It also therefore passes into the condition of plus-systole.

We thus reach the conclusion that in the reaction state, heart, abdominal blood lake, peripheral blood lake, and pulmonary blood lake move together, becoming more definitely systolic.

They likewise, as we have seen, move together towards *diastole* in the rest state.

Their progress into the *systolic condition* is a phenomenon of the sympathetic nervous system and of the action of adrenalin.

A close Connection exists between the Blood Lakes and the Heart.—This is evident, if the foregoing has been sufficiently explicit. The structures react together, they are subject to the same mechanism, both nervous and chemical and physical, they serve in a measure the same purpose in the reaction state, the driving of blood at high pressure into the muscles and brain and the keeping of it there by preventing venous return from the periphery.

The Blood Lakes and the Heart have a close Embryological Relationship. — Gaskell has shown that a strong case can be made out for the view that the heart muscle was originally derived from the so-called "dermal" musculature. He therefore views the heart as a *sphincter muscle,* belonging to the same group as the sphincters of the bowel, *e.g.*, pylorus, and he has been at pains to show that the organ is innervated like the dermal musculature and the sphincters—*i.e.*, it is activated by the sympathetic and by adrenalin. Reasons exist supporting the idea that the arterioles of the skin are of dermal origin, and also the arterioles of the abdominal blood lake. And as Schäfer has shown, these surface arterioles are developed differently from the deeper blood vessels; they arise from what he calls "vaso-formative cells." He thus describes the process :—

"At some parts the tissue is completely studded with these cells, each containing a number of such spheroids and forming groups or 'nests' of blood corpuscles or minute 'blood islands.' The cells become elongated and pointed at their ends, and send out processes to unite with neighbouring cells. At the same time the vacuoles in their interior become enlarged and coalesce to form a cavity within the cell in which the reddish globules, which are now becoming disc-shaped, are found. Finally, the cavity extends through the cell processes into those of neighbouring cells and into those sent out from pre-existing capillaries, but a more or less extensive capillary network is often found long before the connection with the rest of the vascular system is established."

These vaso-formative cells have been found *subcutaneously* and also in the *great omentum* of the cat and rabbit by Ranvier and of the dog by Hayein ; in the *mesentery* of the guinea-pig by Nicolaides ; in the *liver* of the sheep embryo by Kuborn.

This separate development of vessels in the skin and mesentery, etc., that is, *in the blood lakes*, is the more interesting when we recollect that the action of adrenalin is chiefly on these, and scarcely at all, if at all, on the systemic vessels, which, indeed, it is said to dilate. We must also in this connection remember that while adrenalin constricted the limb with the skin on it, it dilated the limb which had been skinned.

The blood lake of the entire skin of the body, then, the blood lake of the abdomen, the blood lake of the lung, and the heart are structures bearing a close relationship to one another and performing in a sense the same function,

The Vasomotor System is much more than a Servant of the Heart or Brain.—The conception of the vasomotor system as a kind of servant of the heart or brain is thus seen to be a very one-sided view. If our study of the reaction state has any meaning at all it makes it evident that the vasomotor system—*i.e.*, the sympathetic—is really the telegraph system whereby the reaction state as a whole is produced, intensified, diminished, or stopped. Thus the heart does not beat faster because the periphery is constricted—but both the fast beat and the peripheral constriction are simultaneous events ordered centrally and mutually, supporting one another towards the accomplishment of a set purpose.

There are not Two Hearts, but Five.—In fact, the blood lakes are "hearts" in the true sense of that term—that is to say, they are propellers of blood, and their activities are mutually interdependent. The heart clearly could not of itself produce the pressure required in the reaction state. It could not by any process of *vis a tergo* drive the mass of the blood from the abdominal lake into the portal system and so close the venous cistern of Keith and rush this body of blood through the lungs. It could not, as contraction of the periphery does, fill up the deep veins and (venous return being impeded by the rush of portal blood into Keith's cistern) thus flood the muscles from behind as well as from before. What it can do is to keep the arteries pumped full, and to play its part in the much greater pumping process in which in a sense the whole body is concerned—the process which gets its great driving force not from the heart at all, but from the recti muscles and the whole musculature of the trunk.

Failure, therefore, of the so-called vasomotor mechanism of the blood lakes, abdominal or mesenteric or pulmonary, must be every whit as serious occurrences as failure of the heart. Their failure, just as much as failure of the heart, will prevent the due accomplishment of the reaction state.

And failure to accomplish the reaction state is most assuredly failure to live.

CHAPTER XII

The Law of the Intestine

HAVING now differentiated certain structures as sphincters—the heart, the peripheral blood lake, the abdominal blood lake, and the pulmonary blood lake—and having discussed the various rôles played by these structures in the reaction state, we must consider the nature of the vessels which lie between them and unite them with one another so as to give a complete circulatory mechanism, and which, in the reaction state, remain in a state not of activity, but of inhibition.

The best approach to this work is a study of the "Law of the Intestine" propounded by Bayliss and Starling, and of the mechanism of intestinal peristalsis propounded by Keith.

The law of the intestine is as follows: "When a peristaltic contraction is caused by stimulation at a spot, the contraction commences above the spot stimulated, and is accompanied by a relaxation of the intestine below the spot."

This law holds good when all the nerves to the gut from the central nervous system have been severed. The mechanism is therefore an end-mechanism, existing in the gut itself.

Keith (*Proceedings of the Royal Society of Medicine*, vol. ix. No. 1, Section of "Electro-therapeutics," p. 4) described so-called neuromuscular sections in the alimentary canal, each provided with a pace-maker like the heart, and each acting by a series of sphincter muscles with peristaltic areas intervening between them:—

"Each section is cut off from its neighbour by a sphincter or sphincteric track which effectively blocks the passage of contraction waves and prevents them spreading from one section to the next. I presume that each section is provided with a special centre or pace-maker, where the chief contraction impulses arise, but I have only succeeded in demonstrating anatomical centres in four of them. I presume that each centre has its own rhythm."

The Work upon the Bowel Mechanism was begun with the definite object of translating Cardiac Physiology to the Musculature of the Alimentary Tract.—This statement was made by Keith concerning Alvarey's work with Cannon at Harvard. The result is thus described by Keith:—

"We have both likened the alimentary tract to a railroad divided into sections, each provided with its signalmen and telephone apparatus. The signalman of one section refuses to accept any further traffic until his section is clear; all the sections are closely correlated; if one is blocked, the others, too, become automatically blocked. . . . Each section of the alimentary tract ends at a sphincter."

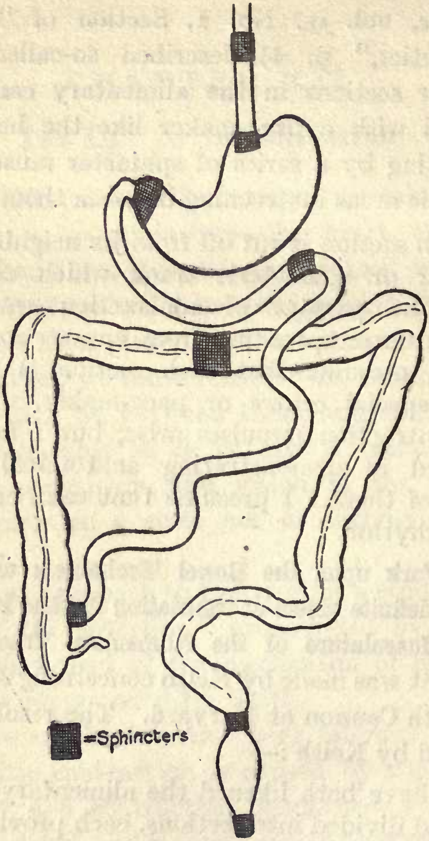


FIG. 45.—(After Keith).

These sphincters are shown in Fig. 45, which is strictly diagrammatic.

Keith further traced fibres of the vagus to various nodal points or pace-makers of the peristaltic areas, and Gaskell showed that the vagus was the motor-nerve to the bowel—peristalsis was a vagal phenomenon.

But it is well known that adrenalin inhibits the bowel peristalsis—this action is used as a test for adrenalin. Further, adrenalin contracts the bowel sphincters (Schäfer), driving them, if the expression may be used, into systole.

Thus adrenalin inhibits peristaltic areas of bowel, and contracts sphincters of the bowel.

This must occur during the reaction state when those other sphincters, the heart and the blood lakes, are being contracted also by adrenalin.

Thus we reach the question : Are there in the vascular system, lying between and connecting the blood lakes or sphincters, peristaltic areas which are inhibited by adrenalin, just as there are in the bowel lying between the sphincters peristaltic areas which are inhibited by adrenalin ?

Evidence that the Systemic Arteries and Veins constitute Peristaltic Areas similar to the Peristaltic Areas in the Bowel.—A study of the pulse reveals the fact that the explanations usually given of the pulse beat cannot be the correct explanations. The usual explanation is that of a tube full of water ; some water is added at one end of the tube, and so some water falls out of the other end. A wave of propagation is thus set up, a form wave of the vessel wall, not a wave corresponding to fluid moved (Fig. 46). On squeezing the ball an amount of fluid equal to the capacity of the ball will fall out of the tube, and a wave will at the same time pass along the tube and be felt by the finger (X) if that be pressed on the tube.

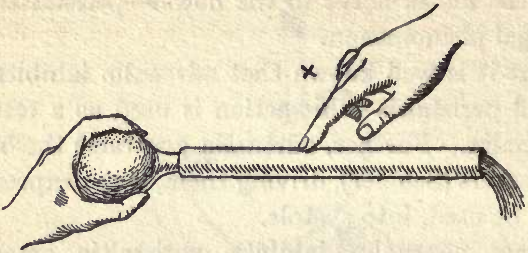


FIG. 46.

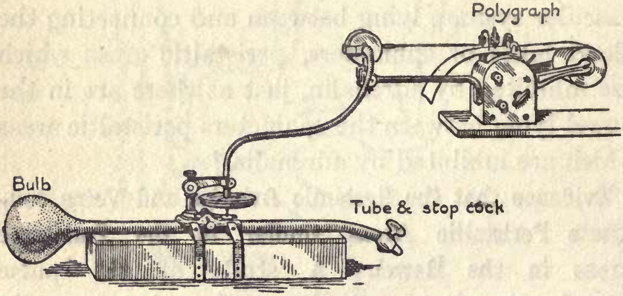
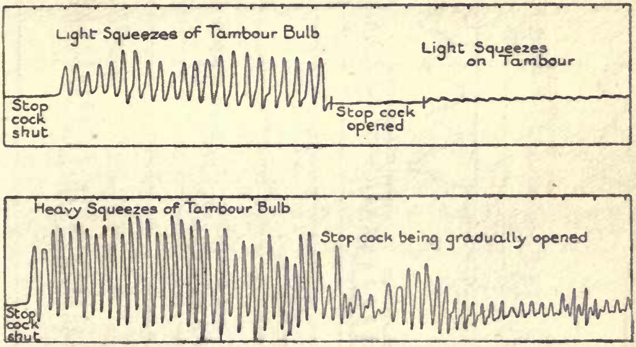


FIG. 47.

If this explanation were correct it would follow that during *peripheral vaso-dilatation* the size of the pulse wave would be diminished to vanishing point, while, on the contrary, during peripheral construction the height of the pulse wave would instantly be increased. That this is so can be proved by taking a rubber tube and partially closing one end of it. The unclosed end is fitted to a ball syringe and connected with the polygraph in such a way that the pulse tambour of the polygraph rests on the tube just as it normally rests on the radial artery. This is illustrated in Fig. 47.

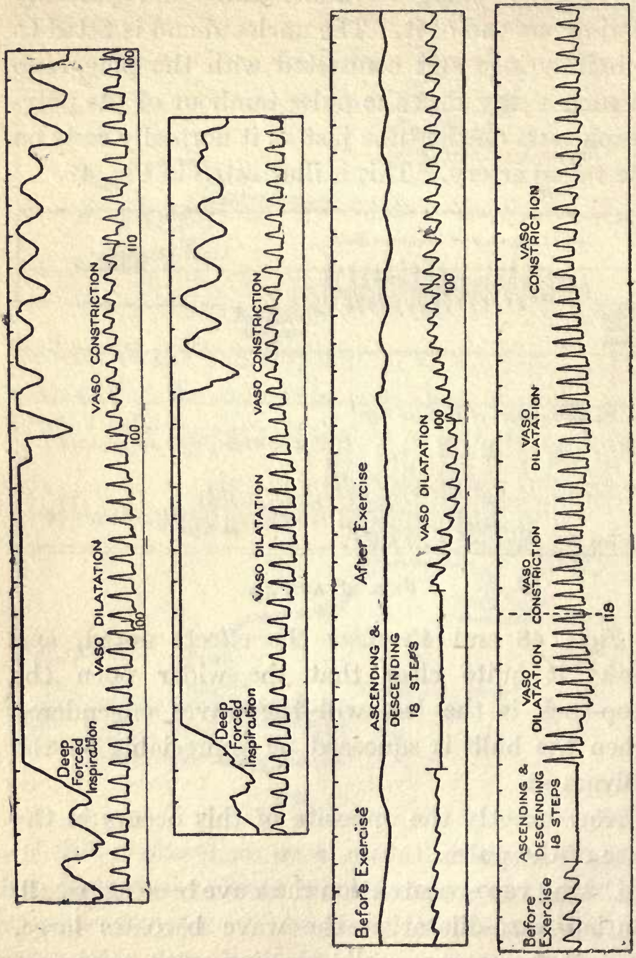


FIGS. 48 AND 49.

Figs. 48 and 49 show the effects noted, and make it quite clear that the wider open the stop-cock is the less will the waves engendered when the bulb is squeezed be appreciable by the polygraph.

Now exactly the opposite of this occurs in the case of the pulse.

During vaso-constriction the wave becomes small. During vaso-dilatation the wave becomes large. And that we are really dealing with vaso-constriction and vaso-dilatation at these times can be proved—quite apart from the look of the patient—



FIGS. 50, 51, 52, AND 53.

by observing how the abscissa line of the tracing rises during constriction and falls during dilatation. This is well shown in Figs. 50, 51, 52, 53, and 54.

It is clear, then, that if with the "stop-cock" open we have not a diminished but an increased size of wave, some other mechanism as well as the heart beat must be at work. *We are forced by a process of exclusion to believe that synchronously with every heart beat there occurs also a beat or systole of the whole peripheral sphincter, whether that sphincter be in a state of plus-systole or plus-diastole.*

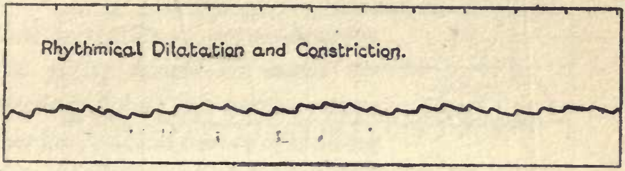


FIG. 54.

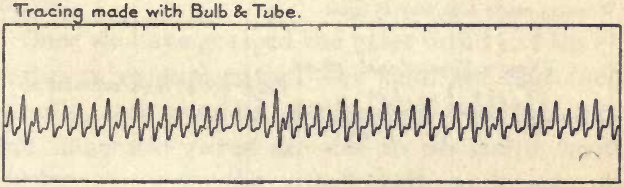


FIG. 55.

In Fig. 55 is shown a curve made by means of the tube and polygraph apparatus figured in Fig. 47. In this case at each stroke of the bulb a finger was placed for an instant on the free mouth of the tube (which was the same length as the radial artery is distant from the heart and of soft consistence). The result is astonishingly like certain pulse tracings—cf. Fig. 56, a real pulse tracing.

In Fig. 57 is shown the same, followed by a period when the finger was not placed on the end of the tube at each stroke of the bulb.

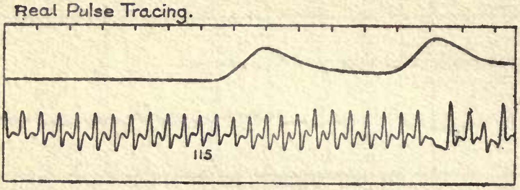


FIG. 56.

This explanation is interesting in view of the facts we have already elicited concerning the presence of the blood lakes, their actions like hearts, and their response to adrenalin. It explains at once why the wave falls more slowly and incompletely to the abscissa in vaso-constriction and why it falls so very completely in vaso-dilatation, while at the same time going up in each instance to its normal height.

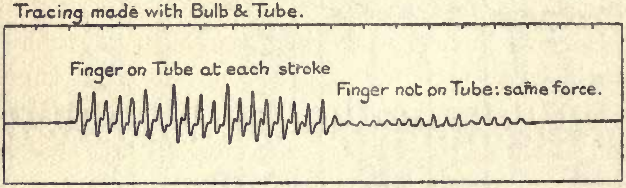


FIG. 57.

It does not, however, explain in any way the dicrotic wave, or, what is more pertinent still, the anacrotic wave.

If we cannot have a pulse wave except with a closed peripheral heart or sphincter, it is very manifest that when that sphincter is open—between the pulse beats—no small reflection from the aortic valves has the remotest chance of making any kind of wave in the radial pulse. Further, even if it had, such an explanation could not by any stretch of the imagination be made to cover anacrotic waves. We are, therefore, in face of our experiments, forced to look elsewhere for explanation of dicrotic waves than the recoil from the aortic valves.

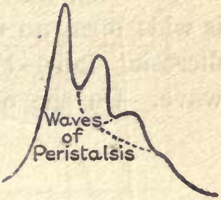


FIG. 58.

Once we have grasped the utter futility of that—aortic valve explanation—we shall see that there is only one possible explanation — the dicrotic and anacrotic waves are due to peristaltic waves in the vessel wall, and are appreciated by the tambour of the polygraph because the tambour is pressing on the vessel wall.

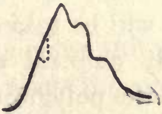


FIG. 59.

In Figs. 58 and 59 the waves of peristalsis are shown.

It is manifest that if this peristalsis corresponds to the peristalsis seen in the bowel it will be most evident in the rest state, and will be less so in the reaction state. Further, that during excessive manifestations of the rest state (*e.g.*, when the reaction state has just passed off) the wave will be very strong, but because the peripheral stop-cock is wide open no dicrotic wave will follow the predicrotic fall — that is, no *perceptible* dicrotic wave. On the other hand, during quiet periods of the rest state, when vaso-dilatation is not excessive, the dicrotic wave will be high up the tracing because the peristaltic wave will be taking place in a vessel not so completely emptied as to be unable to affect the tambour of the polygraph after the wave of peristalsis has gone by.

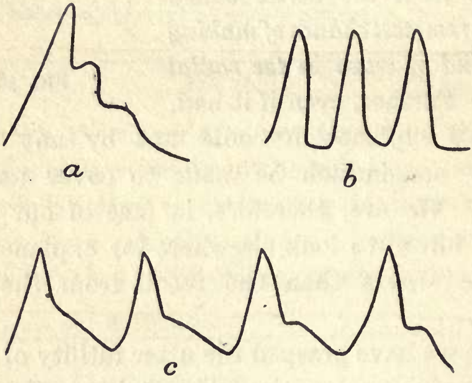


FIG. 60.

In Fig. 60 we see—*a*, Rest state pulse with little vaso-dilatation ; *b*, rest state pulse during marked vaso-dilatation ; *c*, reaction state pulse. The dicrotic wave appears to be quite absent in *b*, and to be very poorly marked in *c*. The reason clearly is that in *b* the excessive vaso-dilatation empties the vessel during the first peristaltic wave after the blood wave. There is no blood left to make a dicrotic wave. In *c* the small and atypical character of the dicrotic wave is due to the strong inhibition of peristalsis which is then occurring.

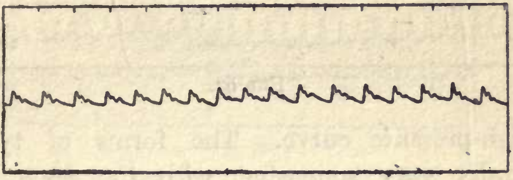


FIG. 61.

Fig. 61 shows a type *a* pulse; Fig. 62 a type *b* pulse—after an injection of apocodeine, which inhibits the whole adrenalin sympathetic mechanism, and so acts against the reaction state; Fig. 63 shows a type *c* pulse in a patient reacting to malaria fever. In this last tracing a period of vaso-dilatation is given—type *b* pulse—which shows how sharply

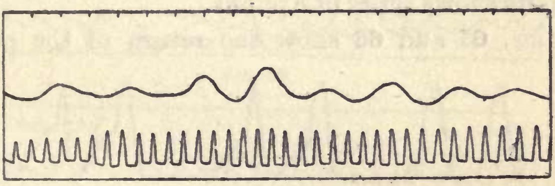


FIG. 62.

the descending arm of the curve is brought down to the abscissa by the peristaltic wave—this is not observed in the type *c* curve, which is, of course, a high-pressure curve. The forms of type *c* naturally vary somewhat with the pressure or rather with the degree of the reaction state present. The more the reaction state is advanced the more active will be the heart and peripheral sphincter, and so the more strongly inhibited will peristalsis be. The less the reaction state the more will the pre-dicrotic or peristaltic wave be evidenced. Fig. 64 shows types of *c* pulses.

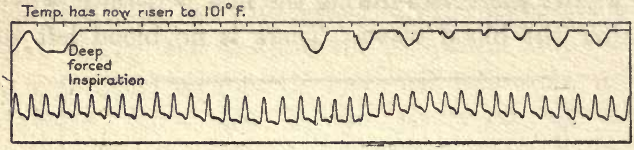


FIG. 63.

Figs. 65 and 66 show the return of the pulse after exercise. Figs. 67 and 68 show the effect of brief effort made during the course of the tracings, and the shading of type *c* pulse into type *a*. Figs. 69 and 70 show the same patient at rest and in reaction.

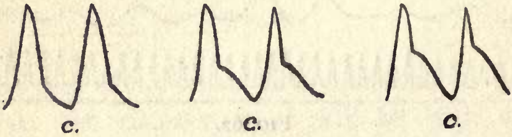


FIG. 64.

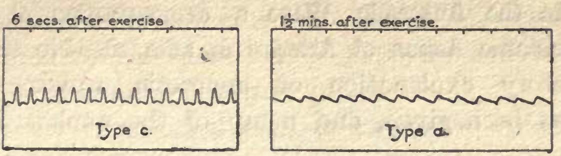


FIG. 65.

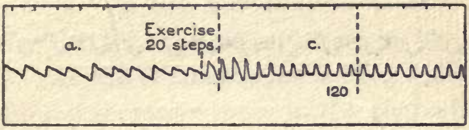


FIG. 66.

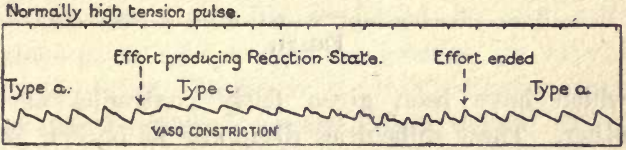


FIG. 67.

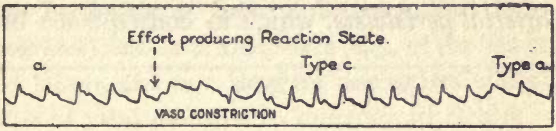


FIG. 68.

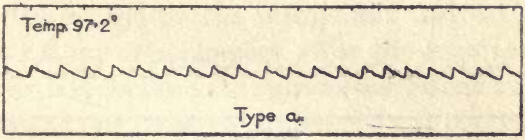


FIG. 69.

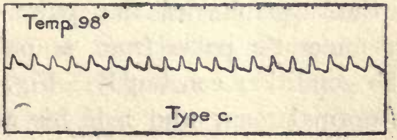


FIG. 70.

In the Anacrotic Wave a demonstration of the Peristaltic Action of Arteries is afforded.—No satisfactory explanation of anacrotic waves has ever been given, and many of the explanations which have been given flatly contradict each other. These difficulties disappear at once if we view the anacrotic wave as an *uninhibited wave of arterial peristalsis*, which in consequence breaks in upon the upstroke of the pulse. Fig. 71 shows an anacrotic pulse from a patient who showed the condition constantly. Fig. 72 shows it from a normal man, who held his arm in the air while the tracing was being taken—this latter

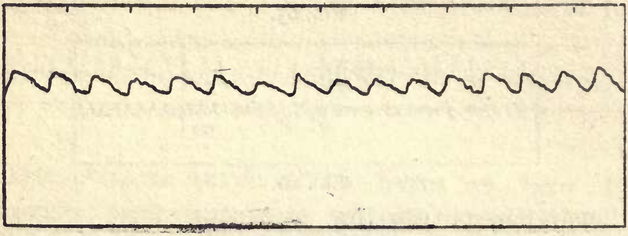


FIG. 71.

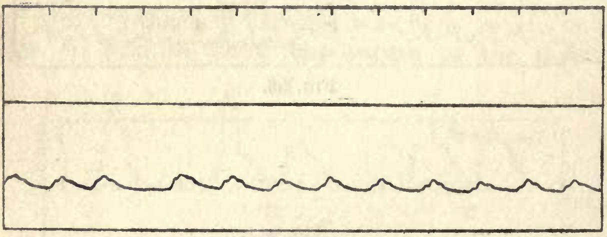


FIG. 72.

manœuvre, as was noted by Von Kries, tending to produce the condition, perhaps by weakening the local inhibition of peristalsis.

Certain Physiological Findings suggest the Peristalsis of Arteries.—Luciani points out (*Physiology*, vol. i. p. 275) that "at the end of diastole the velocity is greater in the peripheral than in the central arteries. At the commencement of systole, on the other hand, it increases more in the central than in the peripheral arteries."

This could not be explained by any other assumption except that the arteries are capable of peristalsis. Moreover, the rich muscular supply of the peripheral arteries adds weight to the assumption.

The Peristalsis of Arteries differs entirely from the Systole and Diastole of Sphincters (e.g., of the hearts and blood lakes), and is a separate mechanism altogether.—It is of vital moment to understand clearly what is meant by the peristaltic waves in arteries and what is meant by the systole and diastole of the blood lakes or sphincters. For the reasons given above, it is assumed that just as the vascular system comes at one point to the heart so at other points it comes to other hearts. These hearts are like sphincter muscles enwrapping the blood tube (Fig. 73), just as the bowel sphincters enwrap the bowel tube (Fig. 74).

Gaskell had this in his mind when he wrote: "Do the cardiac nerve cells" (vagus) "supply motor nerves to any muscle? Is there any evidence of two kinds of muscle in the heart similar to the evidence I have given of two kinds of muscle in the gut? There certainly is evidence, and of a most striking kind. Fano in 1900 found that when records were taken of the contractions of the auricle and ventricle of the water tortoise, *Emys europæa*, a clamp being placed at the auriculo-ventricular groove, the base line of the auricular tracing was not straight but slowly undulating, the rhythm of the waves being often remarkably regular. The auricle of this tortoise shows the presence of two rhythmical activities going on simultaneously, the one a tonic rhythm, having the appearance of Traube-Hering curves, and the other the ordinary heart-beats superimposed on this tonic rhythm.

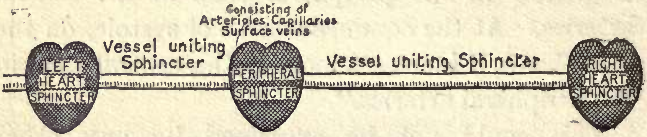


FIG. 73.

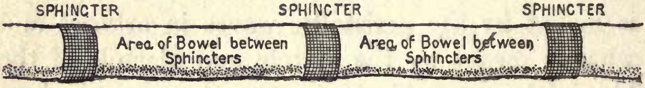

SPHINCTER
SPHINCTER
SPHINCTER
Area of Bowel between Sphincters
Area of Bowel between Sphincters

FIG. 74.

"Fano at first looked upon this phenomenon as indicating the presence of two kinds of muscle in the auricle, the one the ordinary quickly contracting cardiac muscle and the other a more slowly contracting kind, which in consequence of the clamp in the auriculo-ventricular groove was set into rhythmic action. He drew attention to the fact that atropine removed these rhythmic tonic contractions while the heart beats continued, but muscarine removed the heart beats while the tonic rhythm continued. . . . Bottazzi found the two kinds of contraction behaved differently to stimulation of cardiac nerves: stimulation *of the augmentors inhibits these tonic contractions*, so that the auricular beats now all start from the same base line, while, of course, the beats are themselves increased in strength; conversely, stimulation of the vagus will make the tonic rhythm appear, if it is not present, and increase the strength of the waves if they are weak, while, as is well known, it diminishes the strength of the auricular beats. . . . Bottazzi also, in conjunction with Canfini, has examined histologically the auricular tissue of *Emys* and has confirmed Rosenweig's discovery of unstriped muscle fibre in the auricle. They form a strong layer next to the endothelium and continue into the sinus venosus and the beginnings of the great veins; the striated muscle fibres also continue into such a vein as the vena hepatica, always lying external to the layer of unstriped muscle. In agreement with its histology the sinus venosus exhibits a well-marked tonic rhythm which is influenced by the cardiac nerves in the same way as is the auricle.

"These observations definitely prove the existence in the heart of a system of unstriped musculature in addition to the striated cardiac musculature which is especially well developed in the water tortoises, and extends from the auricles into the great veins. This unstriped muscle resembles the enteral unstriped muscles, the bronchial muscles, and those of the gall bladder in its behaviour to poisons like atropine and muscarine, and in its innervation; for all these muscles are supplied with motor fibres from cells connected with the vagus outflow, and with inhibiting fibres from cells connected with the thoracico-lumbar" (true sympathetic) "outflow. On the other hand, the striated cardiac muscle resembles the enteral sphincter muscles in that their motor nerve cells are connected with the thoracico-lumbar outflow and their inhibitor cells with the vagus outflow. In fact, exactly the same kind of reciprocal innervation exists in the heart as in the intestine."

Gaskell thus describes a continuous tube passing from the veins *through* the heart as shown in Fig. 73 diagrammatically, the heart being a muscle derived from an entirely different source and thrown round the vessel.

The following description by Schäfer of the endocardium is highly suggestive on the same point:—

"The endocardium forms a complete lining to the whole of the auricular and ventricular cavities, covering the valves, chordæ, tendiniæ, and papillary muscles. It may be compared to the wall of a vessel such as the aorta. . . . It shows in some regions a division into three tunics, tunica intrina, media, and adventitia. . . . The tunica media contains elastic fibres which abuts on the tunica media. The tunica media contains elastic fibres which in parts somewhat resemble the elastic membranes found in the aorta. . . . The tunica media contains in places scattered bundles of non-striped muscle, as first pointed out by Schweigger-Seidal. Outside the tunica media lies the tunica adventitia, in which the elastic fibres are much coarser than in the media, a feature which reminds one of that seen in many vessels."

We thus conceive of the heart as a circular muscle flung round a vessel which is continuous through it, exactly as the sphincters of the bowel are flung round a tube continuous through them.

The Mechanism of Arterioles at the Periphery is exactly similar to that of the Heart.—We have to think of the arterioles at the periphery also as muscular bundles flung round tubes which are continuous through them—*i.e.*, as millions of tiny hearts (Fig. 75). It is notorious that in these peripheral arterioles the muscular elements largely predominate. They are also innervated as the heart is (*i.e.*, shut by adrenalin). The same thing applies, as we shall see below, to the mesenteric arterioles. Curiously enough, anatomical research has discovered in the skate the very mechanism which we conceive to exist—a system of rings flung round the vessels. Describing this, Schäfer writes :—

" A peculiar arrangement of non-striped muscle is found in the mesh-like blood vessels of the skate, in which the muscle, instead of forming a continuous coat, appears as well-defined rings (P. Meyer)."

We thus reach the conception of tubes with Sphincter Muscles flung round them.—Centrally the sphincter muscle is called "the heart"—peripherally it is called the "arterioles." But it is the same type of muscle in both places, performing the same function—*i.e.*, driving blood onwards. It has nothing in common with the tube it surrounds, it is differently innervated and differently derived. When it is active, the tube it surrounds is passive; when it is passive, the tube it surrounds is active—in other words, a continuous peristalsis is always going on in the tube, just as it is always going on in the bowel. But when the sphincter muscles (heart, blood lakes—*i.e.*, arterioles of peri-

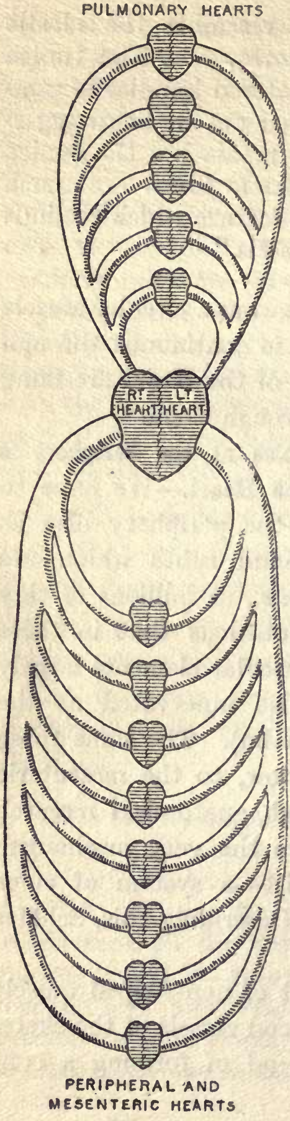


FIG. 75.

phery, mesentery, and lungs) are active and in contraction, the peristalsis in the vessels which they surround is for the moment inhibited: on the contrary, when the sphincter muscles are relaxed the peristalsis in the tubes is active. This is strictly utilitarian, for peristalsis of the tubes against closed and contracted sphincter muscles would be futile. Whereas, when the sphincters are open, peristalsis will fill them up again by hurrying the blood into them.

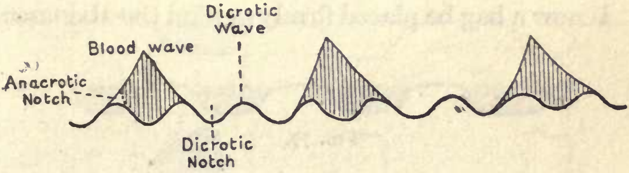


FIG. 76.

Continuous peristalsis of vessel with sphincter waves, or blood waves, or pulse waves superimposed.

The Peristalsis of Vessels which connect the Sphincters or Lakes is therefore a continuous rhythm, having no connection with the rhythm of the heart, but being inhibited during Systole and Systolic States (Reaction State), and being activated during Diastole and Diastolic States (Rest State).—In Fig. 76 is shown the wave of peristalsis with the blood wave or pulse wave or sphincter wave superimposed upon it. It would seem that the wave of peristalsis is much quicker than the sphincter wave, which, however, may be increased or diminished in rate.

The point of real importance is that there are two waves of totally different types superimposed upon one another.

The Mesenteric Blood Lake beats with every beat of the Heart and the Peripheral Blood Lake.—We know as a fact that the two hearts, the right and the left, beat synchronously, thus for the moment shutting the circulation at two places. We have seen very strong reasons for supposing that the peripheral blood lake or sphincter also beats synchronously with the two hearts.

If now a bag be placed firmly around the abdomen and connected with a polygraph, and if, at the same time, the patient be instructed to hold his breath, a tracing will be obtained like that of Fig. 77 (the bag is placed at the level of the umbilicus).

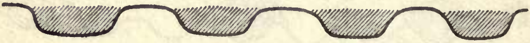


FIG. 77.

This tracing is well enough known, and is usually ascribed to the beating of the aorta in excited subjects. If, however, a little care be taken to analyse the tracing, it will soon be evident that it cannot possibly be due to the beating of the aorta.

If, for example, a tracing of the apex beat is taken simultaneously with the tracing round the waist, it will be found that the relationship is as figured in Fig. 78. Now it is certain that the heart beat cannot begin at the *end* of the aorta beat, which would be an upstroke beat like the pulse beat, but must occur either slightly before or synchronously with it. In Fig. 78, however, a heart beat begins at the down-stroke of *a*—abdominal beat, and, in actual tracings, no less than two-fifths second or more elapses before the next upstroke of *a*—abdominal beat. Now, as it takes only two-fifteenths—one-fifth second—for cardiac systole to reach radial pulse (as shown by tracings), manifestly it cannot take two-fifths second for the cardiac systole to reach the aorta above the navel. *Consequently these beats are not aortic beats.* Nor yet, as might be suggested, are they "reversed" aortic beats, for if a tracing be taken of the aortic beat and, simultaneously, a tracing be taken of these beats, it will be found that the time occupied by the upstroke of the aortic beat is but a fraction of the time occupied by the downstroke of the abdominal wave. The

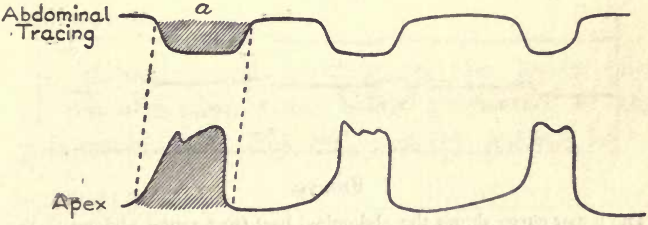


FIG. 78.

abdominal wave is therefore not a "reversed" aortic beat (Fig. 79). If, now, the abdominal tracing be figured in black, as has been done in Fig. 78, it will be seen that these beats correspond exactly in time to a *reversed* cardiac apex tracing.

But if the mesenteric lake goes into systole and diastole like the heart, then at systole the volume of the intestinal content will be less, at diastole it will be greater. That would give a reversed wave to a polygraph, the tambour of which was applied externally to the abdomen. Thus we are able to explain the wave seen exactly.

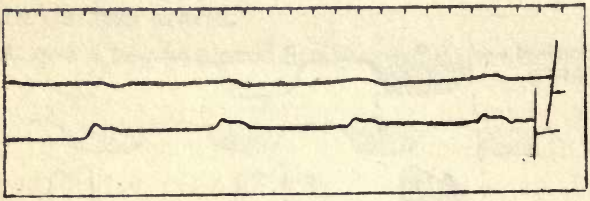


FIG. 79.

The upper curve shows the abdominal beat (bag round abdomen), the lower is the aortic beat. These do not correspond, and are not therefore reversed pictures of one another.

In Fig. 80 is shown the abdominal wall tracing referred to with the apex beat also on the tracing. In Fig. 81 apex and radial are shown. The timing of the events referred to above can thus be measured by the reader. It might, however, be supposed that this "inverted cardiogram" obtained from the abdomen was due to the systole of the heart causing an upward suction of the diaphragm. But this possible source of error can be excluded.

If a tambour is placed on the upper part of the abdomen and another on the lower part of the abdomen, both being connected to the polygraph (as in Fig. 82), and the patient be instructed to hold his breath, the tracing will show the "inverted cardiogram" in both instances—Fig. 83. If now a flat ruler be thrust down firmly edgeways on the abdomen so as to divide the abdomen into two and separate the tambour, it

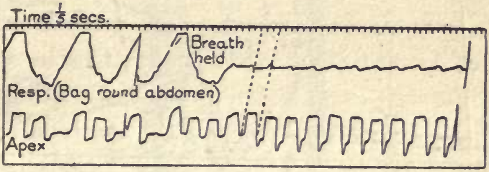


FIG. 80.

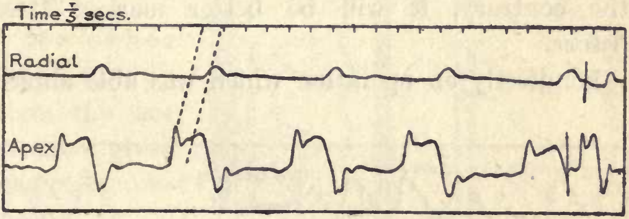


FIG. 81.

will be found that the respiratory wave will be almost abolished in the lower tambour (Fig. 84). But the "inverted cardiogram" or sphincter wave

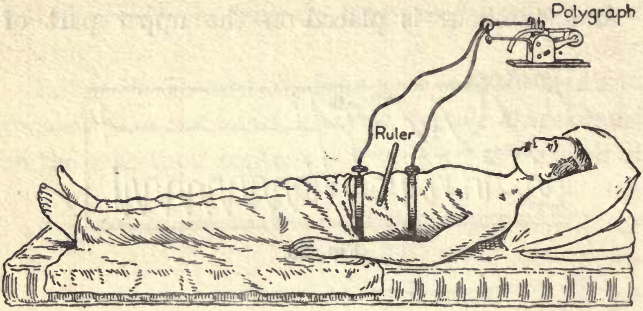


FIG. 82.

will not be abolished in the lower tambour. On the contrary, it will be better marked than before.

Manifestly an operation which was able almost

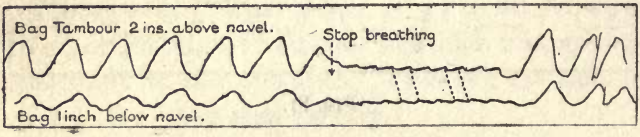


FIG. 83.

to abolish the wave resulting from so great a movement of the diaphragm as the respiratory movement would utterly blot out the very small movement which might result during cardiac systole. Here, however, it is the big movement only which is affected.

Therefore the small movement cannot be due to the cardiac systole acting on the diaphragm. It must lie within the abdomen itself.

No other explanation than the one we have given therefore remains.

This can be further proved by causing the patient to undertake forced expiration with the

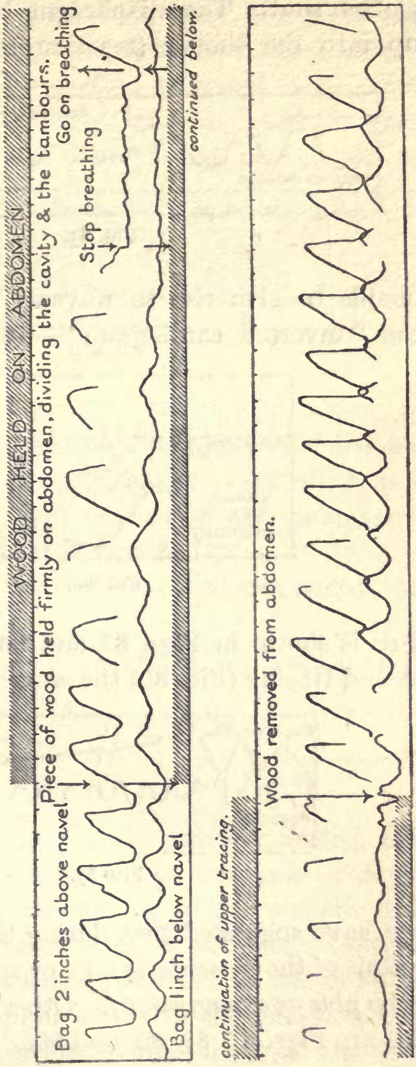


FIG. 84.

glottis shut. The diaphragm being now forced up into the thorax (reaction breathing) will be unable to give rise to waves. Yet the waves—the "inverted cardiogram"—are seen as before.

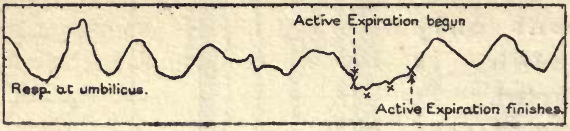


FIG. 85.

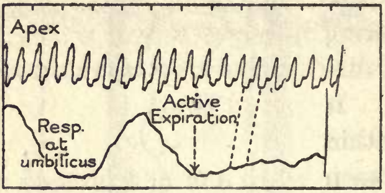


FIG. 86.

This is shown in Figs. 85 and 86. Indeed, in the second tracing (Fig. 86) the waves are exaggerated (we have suggested that during this reaction state systole of the mesenteric lake or sphincter will tend to be *plus* or exaggerated). Other tracings showing this are Figs. 87, 88, 89, and 90.

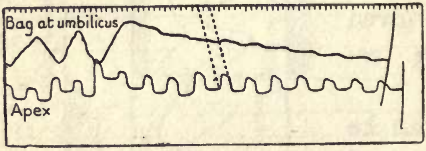


FIG. 87.

The Pulmonary Blood Lake beats like the Heart, Peripheral Blood Lake, and Mesenteric Blood Lake.—We saw that adrenalin exercised less effect on the pulmonary blood lake or sphincter than it exercises on the other blood lakes, and we discussed this fully. We called the action of adrenalin here a tonic one. But systole and diastole would seem to occur here also. We shall now consider whether this beating of the pulmonary sphincter can be proved or not.

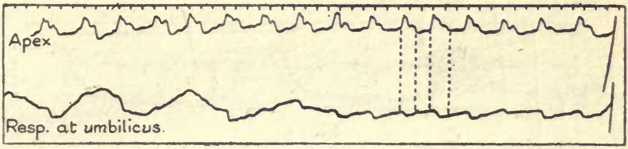


FIG. 88.

If a tracing be taken of the apex beat, and at the same time a tube placed in the patient's nostril, and the patient instructed to stop breathing without at the same time closing his glottis, a curve like that figured in Fig. 91 will be obtained. Actual tracings are shown below (Figs. 92, 93, 94, and 95).

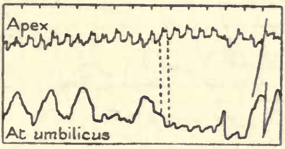


FIG. 89.

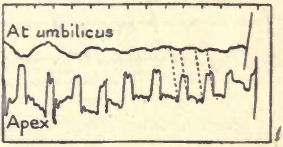


FIG. 90.

Now these reversed waves in the nasal tracing mean a slight *inspiratory* movement of air, and this clearly occurs synchronously with the heart beat. But the suction is not due to the heart beat, because impact of the heart on the lung would

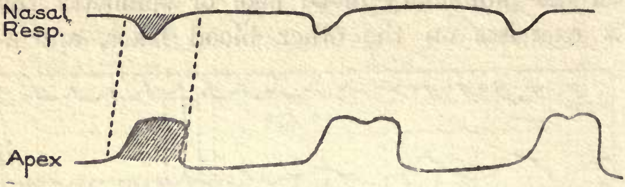


FIG. 91.

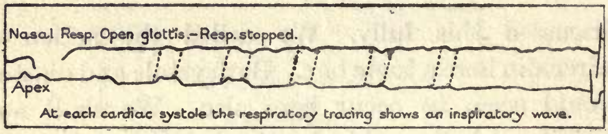


FIG. 92.

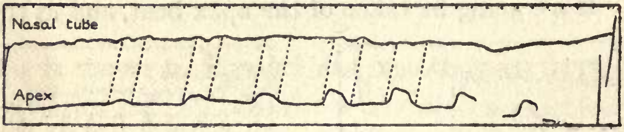


FIG. 93.

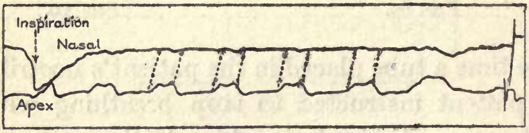


FIG. 94.

expel, not draw in, air; moreover, the blood volume in the chest is not changed—what leaves the heart going into the aorta and what leaves the lungs on one side entering them on the other. Nor does it come from above the glottis, because it disappears *instantly* if the glottis is closed.

The wave, which is a constant (though somewhat difficult to obtain because of the trouble of getting the patient to stop breathing and at the same time keep his glottis open), must therefore be due to a slight systole of the pulmonary blood lake. The inevitable result of such a systole—and so of a reduction of blood volume—would be an increase of air and so increase of air volume. As the heart goes into diastole, so does the pulmonary sphincter, causing then an *expiratory* movement. (This systole and diastole of the pulmonic sphincter can be further shown by a bag tambour placed round the chest: an "inverted cardiogram" is then obtained—Figs. 96 and 97.)

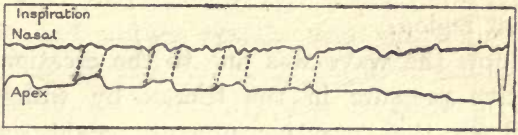


FIG. 95.

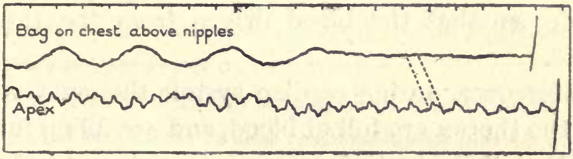


FIG. 96.

This wave has been observed before by Delêpine, and explained as being due to a slight negative pressure made in the thorax by the outflow of blood from the left heart at systole. The wave, as is indicated in the text, can be got also from the thoracic walls and the abdomen at any point in that region.

If now the wave was due to the creation of a negative pressure in the thorax by withdrawal of blood, that negative pressure could scarcely affect the abdomen, since it is largely into the abdomen that the blood driven from the thorax goes.

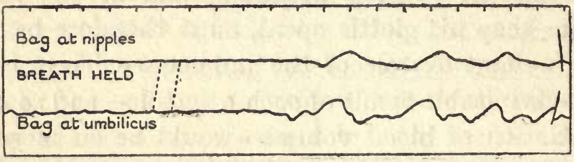


Fig. 97.

Moreover, during cardiac systole the great veins in the thorax are full of blood, and are filling fuller, so that this negative pressure consists only of the difference in volume between the blood arriving by the great veins and the blood leaving by the aorta—it is not reasonable to suppose that this difference would be sufficient to cause an indrawing of the whole thoracic wall, an inrush of air by the mouth, etc.

Further, the wave is a fall, followed by a definite rise, sometimes greater than the fall. Now if it was merely due to the negative thoracic pressure, it would merely be a fall, and there would be no rise again, or at least there would be no rise until the very end of diastole, when the heart was full. The fall and the rise occur, however, in systole and during systole, and this cannot be explained by any theory of negative pressure in the thorax, for manifestly that negative pressure would rise to a maximum at the end of systole, would not have disappeared entirely by the end of systole, as the tracing—accepting this explanation—shows.

We may *therefore conclude* that the theory of negative pressure in systole will not meet the facts of the case. The idea of a systole of the pulmonary sphincter or heart will meet the case and meet it exactly.

NOTE.—The reader is referred to the Preface for a further discussion of this point.

There are therefore five hearts, all of which beat at the same rate and in the closest relationship to one another. They are connected by peristaltic areas, the peristaltic waves in which are inhibited during systole of the hearts, and are activated during diastole of the hearts.—This brings us to the conclusion that the mechanism of the cardio-vascular system is exactly similar to that of the alimentary system, and that in reaction states, as we noted by a totally different line of observation, the sphincters or hearts of the cardio-vascular system go into plus-systole—just as the sphincters of the bowel do (*e.g.*, effect of adrenalin on the ileo-cæcal valve). In the rest state the sphincters pass into plus-diastole, and the peristaltic areas are activated.

CHAPTER XIII

THE VAGUS—NERVE OF DIASTOLE

THE vagus has been described by Luciani and other physiologists as "the Nerve of Diastole." By this is meant that vagus stimulation slows the heart and holds that organ for a time in diastole, thus augmenting the diastolic phase at the expense of the systolic. This action is well illustrated in Fig. 39 (p. 67), where occasional long beats are seen to occur.

The vagus, therefore, so far as the heart is concerned, directly opposes the action of the adrenalin or true sympathetic, and, so far as the heart is concerned, is, clearly, the nerve of the rest state just as the true sympathetic is the nerve of the reaction state.

But its effect upon the heart is only a small portion of the activity of that great sympathetic system which is called now the vago-sacral sympathetic system, and now the bulbo-sacral sympathetic system—these names being designed to differentiate this system from the true sympathetic or adrenalin sympathetic.

When we speak of the vagus, then, we mean not only, not even specially, the nerve which runs to the heart—we mean the whole vagus sympathetic picture with all the manifold effects which that vagus-sympathetic certainly exercises. In fact the vagus is only one branch of a system: it would be better to speak of the system as the dilator or diastolic system and not as the "vagus system."

The Vagus, as the Nerve of Diastole, actively inhibits the Heart Sphincter.—We need not stay to discuss this, as it is universally admitted. What we must do is to point out the real meaning of this phenomenon. If the vagus produces plus-diastole in the heart, just as the adrenalin sympathetic produces plus-systole, then *the vagus is a dilator of one of the series of "sphincters" which we have suggested are a feature of the cardio-vascular system* (just as its own sphincters are a feature of the bowel system). These sphincters—the two hearts, the abdominal blood lake, the peripheral blood lake, and the mesenteric blood lake—*are similarly innervated by the adrenalin sympathetic,* and go into systole *and diastole* together.

The presumption, then, that the vagus or dilator system of sympathetic nerves, which is known to inhibit two of the sphincters, *i.e.* right and left heart, inhibits all these others is a strong one. It is supported by the available evidence so far as that goes.

The difficulty about the evidence, however, is that the physiological work has always been done upon the vagus nerve itself—that is to say, upon one branch of the dilator system. Thus we know that stimulation of the cardiac vagus inhibits the heart, stimulation of branches to the lungs contract the bronchioles (adrenalin opens these), stimulation of abdominal branches causes peristalsis and opens the bowel sphincters, and so on (which again adrenalin opposes). We desire, however, to deal not with one branch, the vagus, but with the whole vagus sympathetic system.

The Depressor Nerve is the Key to the whole Vagus Sympathetic System.—This nerve has attracted far too little attention. It is a most remarkable structure. It arises in the aorta, and passes from there to the centres in the bulb, connecting among others with the vagus centre. It is wholly and entirely an afferent nerve. Cut it and stimulate the end in contact with the aorta—nothing happens. But cut it and stimulate the end in contact with the bulb—

You get a Vaso-dilatation of the whole body, and you get Vagus Action.—The effect is thus described by Schäfer :—

" On excitation of the central end of this nerve Ludwig and Cyon witnessed a surprising fall of arterial pressure accompanied by a slight decrease in cardiac frequency . . . on repeating the excitation of the central end after section of both vagi, the arterial pressure fell to an undiminished extent, but the pulse-rate, in this case, remained unchanged. After section of all the cardiac nerves the effect was still the same. This nerve was named by Ludwig and Cyon the depressor, for it possessed the power of depressing the arterial tension by 30 to 50 per cent.

"Ludwig and Cyon observed that on stimulating the depressor *the kidney flushed red with blood*, while the fall of arterial tension became insignificant after section of the splanchnic nerves.

". . . Nor does the depressor act while the arteries are contracted by intravenous injection of suprarenal extract.

". . . According to Bayliss, section of the spinal cord at the level of the second lumbar nerve-root prevents, when the depressor is excited, any manifestation of dilatation in the lower limbs, and fibres producing vaso-dilatation of the legs have been found by Bradford and Bayliss. . . .

". . . No other nerve in the body can, on excitation of the central end, uniformly produce a reflex full of blood pressure. *Almost all parts of the vascular system can be thrown into dilatation by the depressor nerve . . . nevertheless the splanchnic area is by far the most important seat of dilatation . . . the splanchnic floodgate can be thrown rapidly open thereby, and the peripheral resistance lowered.*"

Here, then, is a mechanism closely allied to the vagus system whereby the peripheral and mesenteric sphincters are thrown open.

This Mechanism being placed in the Aorta or in the Heart will be activated during periods of high tension—that is, during the Reaction State.—The depressor nerve is thus a link between the reaction state and the rest state, and its effect will be to temper the reaction state with the rest state when the former tends to become too severe.

Admittedly this evidence that the Vagus or Dilator Mechanism opposes the Adrenal Sympathetic Mechanism by inhibiting all Sphincters is not yet complete.—We cannot speak in advance of experiment, but we can fairly set down the following facts :—

1. The five sphincters (heart and blood lakes) are activated by the adrenalin sympathetic.
2. They can be shown to beat synchronously, and to go into plus-systole and plus-diastole synchronously.
3. In systole they affect the reaction state and in diastole the rest state.
4. The vagus or dilator sympathetic is found to inhibit certain sphincters—the bowel sphincters—which are activated by the adrenalin sympathetic.
5. The vagus or dilator sympathetic is found to inhibit two of the five cardio-vascular sphincters—the two hearts.
6. A mechanism has been found in connection with the dilator and vagus centres which inhibits all the other sphincters—the depressor nerve—while at the same time inhibiting the heart through the vagus.

It is a reasonable conclusion that this mechanism represents one mechanism comprehending the vagus and other nerves—the so-called vagus sympathetic—and that it acts as an opponent of the adrenalin sympathetic by inhibiting sphincters and blood lakes, and so opening these, emptying the muscles and inducing the rest state.

Further, the depressor nerve acts as a link between the adrenalin sympathetic mechanism and the vagus or dilator sympathetic mechanism, which latter, indeed, seems to correspond to the "governor" on an engine.

The Vagus or Dilator Sympathetic Mechanism not only inhibits Sphincters in the Cardio-Vascular System, it has an active role as well, and activates Peristalsis in Systemic Vessels.—We were able to show that during the reaction state the cardio-vascular sphincters (heart and blood lakes) went into plus-systole, while the peristalsis of vessels, as evidenced by the appearance of the pulse wave, was inhibited to a large extent—*i.e.*, the katachrotic waves disappeared or became much less well marked.

We have raised a strong presumption that, opposing this mechanism, the vagus or dilator sympathetic, in the rest state, inhibits and opens the cardio-vascular sphincters. Does it complete the picture by increasing peristalsis in the peristaltic areas of vessels lying between the sphincters ?

This is its effect in the bowel. Further, as we have seen, the pulse in the rest state, provided that active vaso-dilatation is not in progress, always shows katachrotic waves high up the descending arm of the tracing (Fig. 98). (During active vaso-dilatation, as we pointed out, these waves could not be seen because the blood ran out of the tube so fast that no waves were made by it.) In the reaction state, on the contrary, the katachrotic waves came down the curve, and are often nearly absent (Fig. 99).

Hence we are entitled to conclude that peristalsis of vessels is augmented in the rest state and inhibited in the reaction state.

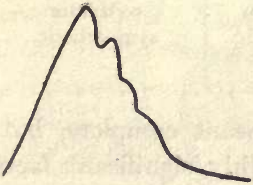


FIG. 98.

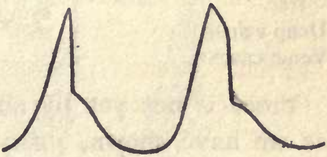


FIG. 99.

But we have been able to raise a strong presumption that the vagus or dilator sympathetic mechanism controls the rest state just as the adrenalin sympathetic mechanism controls the reaction state.

Therefore the vagus sympathetic mechanism when in operation is favourable to the occurrence of increased peristalsis in the vessels joining the sphincters of the cardio-vascular system, just as it is favourable to increased peristalsis in the bowel tracts joining the sphincters in the alimentary system.

The following provisional table expresses the idea we have been following :—

Right heart.	Activated by adrenalin sympathetic.	Inhibited by vagus or dilator sympathetic.
Left heart.		
Mesenteric sphincter or blood lake.		
Peripheral sphincter or blood lake.		
Pulmonary sphincter or blood lake.		
Pulmonary artery.	Inhibited by adrenalin sympathetic.	Activated by vagus or dilator sympathetic.
Pulmonary veins.		
Aorta and arterial tree.		
Deep veins.		
Venæ cavæ.		

Proof is not yet by any means complete, but, as we have shown, many highly significant facts are available. Meanwhile we would emphasize that when we speak of pulmonary blood lake or sphincter we are not including the pulmonary artery or veins which belong to the peristaltic area connecting the sphincters, and not to the sphincters themselves. This is shown diagrammatically in Fig. 100.

The Explanation of the Cardio-Vascular System just given explains certain Phenomena which otherwise are inexplicable.—It is a matter of observation that during the reaction state the patient becomes quickly pale, and then again the pallor passes away, being succeeded by a blush. This pallor is not at all like the pallor seen during

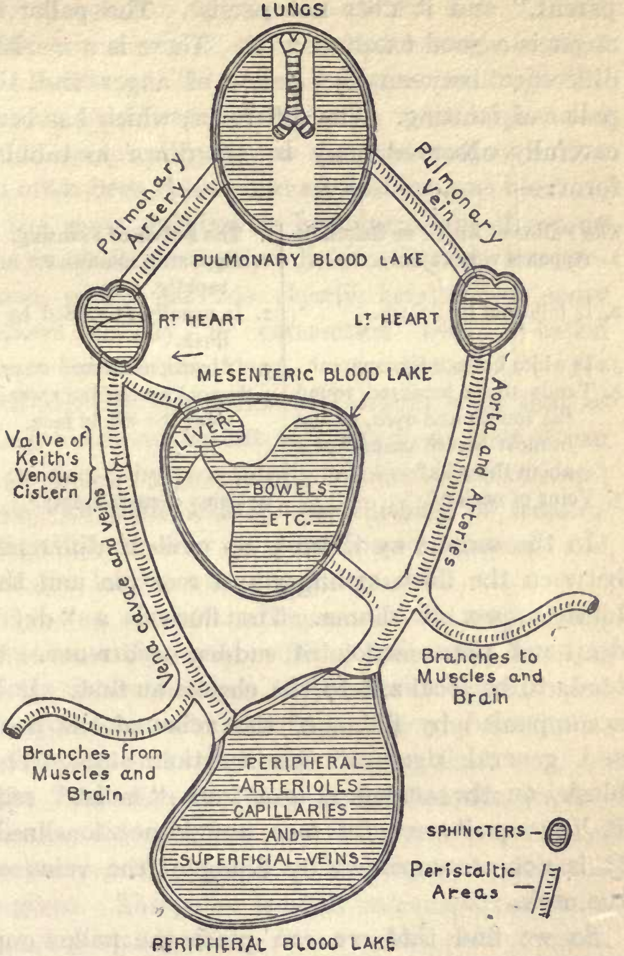

LUNGS
Pulmonary Artery
Pulmonary Vein
PULMONARY BLOOD LAKE
RT HEART
LT HEART
MESENTERIC BLOOD LAKE
LIVER
BOWELS
ETC.
Valve of Keith's Venous Cistern
Vena cavae and veins
Aorta and Arteries
Branches to Muscles and Brain
Branches from Muscles and Brain
PERIPHERAL ARTERIOLES CAPILLARIES AND SUPERFICIAL VEINS
SPHINCTERS
Peristaltic Areas
PERIPHERAL BLOOD LAKE

FIG. 100.

a fainting attack, for it is active, it is not "transparent," and it does not persist. The pallor of anger is a good example of it. There is a marked difference between the pallor of anger and the pallor of fainting. The difference, which has been carefully observed, may be set down in tabular form:—

The Pallor of Anger or Reaction.	**The Pallor of Fainting.**
1. Appears very rapidly.	1. Appears somewhat less rapidly.
2. Is followed by a flush.	2. Is usually preceded by a flush.
3. Is white but not transparent.	3. Is transparent and waxy.
4. Tends to be localized round the mouth and eyes, except in most severe cases "pale about the gills."	4. Is not localized but spreads over the whole face.
5. Veins of neck fill.	5. Veins of neck empty.

In the same way there is an evident difference between the flush of anger and reaction and the blush of sex or shame. The flush is a "dry" deep red phenomenon of sudden occurrence. It tends to be localized to the cheeks at first. It is accompanied by filling of the veins of the neck and general signs of the reaction state. The blush, on the contrary, is a fresh "moist" red. It "sweeps" over the face, and is not localized. It is not accompanied by filling of the veins of the neck.

So we find that we can place the pallor and the flushing of anger in one group as reaction phenomena, and the waxy pallor of fainting and the blush of sex in another group as phenomena not belonging to the reaction state.

The Importance of differentiating between types of Facial Change.—It is evidently exceedingly important to differentiate between these types, and not to accept all pallor as the same pallor—as has too often been done—and all flushing and blushing as the same flushing or blushing. For if we do this we shall inevitably fall into the hopeless confusion which has too clearly befallen so many workers already in connection with so-called "vaso-constriction" and "vaso-dilatation." We shall identify the pallor of reaction with that of collapse, and so be left in amazement that it can be produced by two totally different mechanisms; again, we shall identify the flushing of reaction with that of sex or pure vaso-dilatation, and again be left in utter bewilderment.

The way out of the impasse is the way of observation—the way which leads to a clear differentiation between these various types, based not upon colour alone but upon the whole attitude, appearance, and symptoms of the patient.

The Production of Pallor in the Reaction State.—This can be observed any day, and can always be observed if intravenous injections of adrenalin are given. The pallor is of an untransparent white type of quick duration. It is a sudden phenomenon. During its presence the deep veins of the neck fill, and the whole reaction picture is present with a rapid heart—or a heart becoming rapid—and tense recti muscles.

This pallor is manifestly due to the shutting of the peripheral blood lake and peripheral sphincter, and when it occurs all the great cardio-vascular sphincters are going into plus-systole.

It seems probable that this is the type of pallor which occurs in angina pectoris.

The Production of Flushing in the Reaction State.—The pallor of the reaction state may be followed by a dull red flush, or the pallor may be so evanescent or so very slight that it is not observed, and only the flush is observed—as occurs in hundreds of the small reaction states of everyday life, which fall short of the active emptying of the peripheral lake which pallor signifies.

This flush is usually ascribed to vaso-dilatation following vaso-constriction. *It is, however, nothing of the kind.* While it is seen the reaction state is continuing, the veins of the neck are full, the pulse is rapid, the abdomen muscles are tonic and active, and the glottis is also tonic.

Now, if this type of flushing be carefully observed, it will be seen that, so far as the face is concerned, it tends to be better marked in certain situations and to be badly marked in other situations. This is shown in Fig. 101.

Now it will be remembered that around the mouth and nose the skin is thickest, while over the malar processes it is thin.

"It will be noticed how thin the skin is in the eyelids and how thick round the mouth and side of the nose" (Parsons and Wright, *Anatomy*, vol. i. p. 108).

It is curious, then, that our flush occurs most evidently where the skin is thin, and that it does not occur to the same degree where the skin is thick. (This is well seen in shivering from cold, a reaction state.)

It would seem that the explanation must be that the flushing is a deep flush occurring *under* the skin, *i.e.*, in the vessels which lie outside the skin vessels proper.

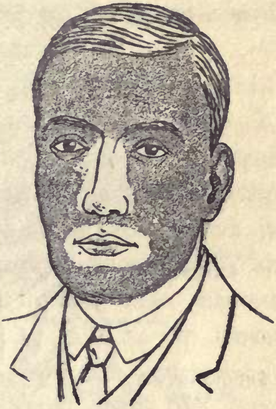


FIG. 101.

There are two small arterial plexuses under the surface of the skin (Fig. 102)—the subpapillary plexus, and, deeper down, the subcutaneous plexus. If, now, we suppose that the peripheral blood lake includes the subpapillary plexus *but not* the subcutaneous plexus, it will be clear that in reaction states the subpapillary plexus will tend to be emptied of blood because the whole peripheral blood lake is being emptied. But the subcutaneous plexus—which is part of the peristaltic area uniting the peripheral blood lake with the heart—

will not be empty. On the contrary, it will be brimful, the high arterial tension being a feature of the reaction state.

The blood in the subcutaneous plexus will, therefore, tend to *shine through* the empty subpapillary plexus wherever the skin is thin. Where the skin is thick this will not occur, and these parts, *e.g.*, the mouth, will remain pale. (Hence, perhaps, the expression " pale about the gills " of persons who are very cold or very upset.)

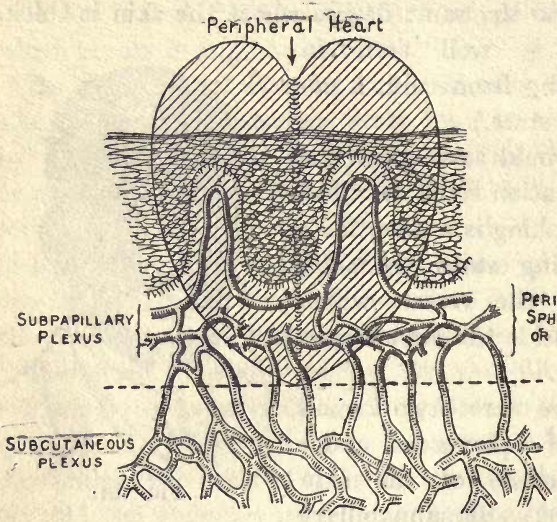


FIG. 102.

The Production of Blushing in the Rest State.—Now in the rest state the peripheral blood lake is in plus-diastole and is full of blood. The arteries, too, are in more active peristalsis—the sphincters being under inhibition. It is quite easy to understand that with the active arteries syringing blood into the open peripheral lake a blush will tend to overspread the face. It is this type of blush which shows occasionally what is called *capillary pulsation*—a blush which waxes and wanes with each cardiac systole, but which, as we have shown above, is not cardiac in origin but peripheral—in other words, we are looking at the systole and diastole of the peripheral sphincter.

The Production of Pallor in the Rest State.—This, as we have seen, is the pallor of collapse. It occurs when the plus-diastole of the great blood lakes is so great that the blood volume in the vessels is reduced to a low ebb. The blood is gathered in the vast abdominal blood lake or heart, and though the skin lake is also dilated there is not enough blood to fill it. It looks waxy and pale.

This explanation throws light upon the slow "vagus" pulse met with in fainting fits. The description of an attack of this sort given by Lewis in his "Report on Soldier's Heart" is worth quoting:—

"A young soldier who presented the characteristic symptoms of tonic debility was sitting at rest in a chair. He began to feel queer as though his 'stomach had turned upside down'; he became dizzy; pallor was noticed. The head fell forward to his knees. He was at once placed in a long easy-chair and further observed: by this time pallor was intense, and he was restless. The pulse was imperceptible, the heart sounds were distant, the rate of beating being 50 per minute; the action was for the most part regular, a single premature beat being noted. From time to time there were retching movements, the pupils were little, if at all, dilated; he was limp, mentally confused or actually unconscious for several minutes. A heavy sweat broke out over the forehead and spread over the chest and body; the pallor remained extreme; respiration was slow and sighing. The pulse was imperceptible for several minutes; as it returned, the systolic blood pressure was registered (palpatory and auscultatory) at 60 mm. Hg. A little later the pressure fell to 55, and then to 50, the pulse ranging in rate between 50 and 60 . . . the average blood pressure of this man in ordinary circumstances was 118, the pulse rate 80–90 . . . the simultaneous fall of pulse rate and blood pressure stamp the attacks as vagal in origin, a view fully confirmed by the occurrence of simultaneous gastric disturbances."

Thus Flushing and Pallor occur in both Reaction and Rest States and by entirely different Mechanisms.—Understanding this point clears up a host of difficulties. It also reveals the fact that in the case of the peripheral blood lake or sphincter particular areas can advance into plus-systole or recede into plus-diastole without affecting the general condition of the blood lake as a whole. This is, of course, well known as a point of observation, *e.g.*, "burning ears," etc. That is to say, that while the blood lake or sphincter beats as a whole according to the general cardiac rhythm, small areas of it may pass into fuller diastole or may remain in more complete systole as the needs of the locality demand. *The peripheral blood lake is thus not only one lake, it is also a large number of small ponds.* The whole body of the lake has its one great ebb and flow (systole and diastole), but each little pond opening into the lake is fitted with sluice gates whereby the local ebb and flow may be increased or may be diminished.

It is impossible to show the Beating of the Peripheral Sphincter or Blood Lake in a Plethysmogram.—This point should be borne in mind. A plethysmogram of the human arm or leg shows only the pulse wave with its peristaltic, katachrotic waves. It does not show the systole and diastole of the sphincter. The reason is that the skin is so thin in comparison with the volume of blood in the limb. The muscles, of course, do not belong to the peripheral sphincter, and the ebb and flow of blood into them is that seen in the main arterial trunks. This picture dominates the plethysmogram because of the huge bulk of muscle present.

CHAPTER XIV

THE ACTION OF PITUITARIN

WE have seen that in the case of the true sympathetic, or adrenalin sympathetic, the auto-drug adrenalin is a specific activator of this nervous system. Where the true sympathetic, when stimulated, produces action, adrenalin produces action, and where the true sympathetic, when stimulated, produces inhibition, adrenalin produces inhibition.

Adrenalin is, therefore, as we saw, a chemical having a *specific* action upon true sympathetic fibres. So that the equation—

a (true sympathetic)$+b$ (adrenalin)$=$C (reaction state) holds good.

But if this holds good what of the rest state ?

Are we entitled to suggest that in the case of the vagus or dilator sympathetic an equation ought also to be produced ? For example, as follows :—

a (vagus sympathetic)$+b$ (?)$=$C (rest state).

We think that we should be so entitled were it possible to point to any other auto-drug the effect of which was the same as the effect of dilator and vagus stimulation.

The Effects of Pituitarin are like the Effects of Dilator or Vagus Sympathetic Stimulation.—A considerable number of subcutaneous injections—though not of intravenous injections—of pituitarin have been given by the writer and the effects closely observed. In all cases where a rapid rate of the heart is observed, followed after exercise by a slow return of the pulse to the normal—indicating a vagus "lag" or slowness of the vagus to pull the pulse back to normal—it will be found that administration of pituitary extract ($\frac{1}{2}$ c.c. intramuscularly daily) will, while the injections are being given, and in the great majority of cases, alter the picture and cause the "vagus grip" to be markedly increased and augmented. This is well shown in the Charts Nos. 1 and 2, Figs. 103 and 104, which were

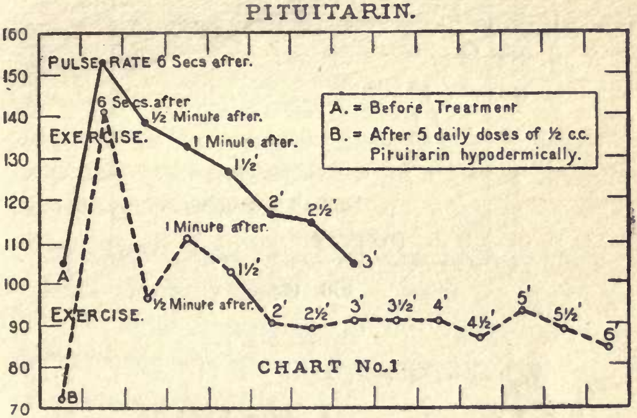


FIG. 103.

selected at random from a series of cases. (It is interesting to note that a very similar effect

PITUITARIN.

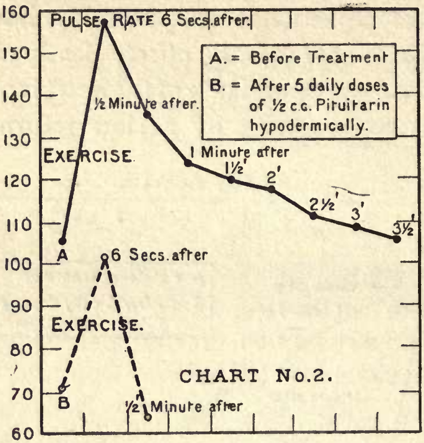


FIG. 104.

CHOLINE

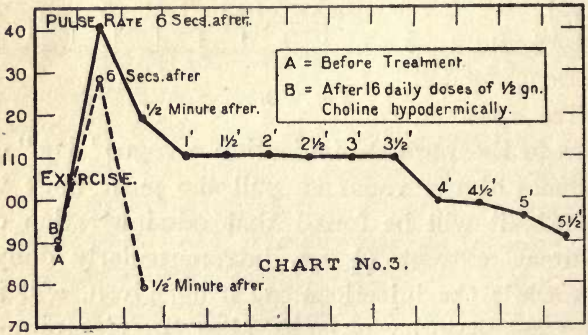


FIG. 105.

was obtained in certain cases with choline given hypodermically—Chart No. 3, Fig. 105.) All these records of pulse rate were taken by polygraph.

That the failure of the pulse to return to normal is due to vagus " lag " seems to be quite certain. Lewis adopts this view in his " Report on Soldier's Heart " to the Medical Research Committee, in which he states :—

> " Yet in its action (in cases of disordered action of the heart) the vagus is not normal. Of this there are several indications. A lag in the recovery of vagal control at the termination of exercise is the rule in the severer cases of the disorder, and is considered responsible for the delay in the return of the previous heart rate."

Accepting this view, we can only explain the effects of pituitarin in the Charts we have given as due to a stimulation of the vagus mechanism giving for the time being a better control. (It was found that when the drug was stopped the patients tended to relapse to their former state.)

The Effect of Pituitarin is not upon the True, or Adrenalin, Sympathetic.—This is a matter of universal physiological finding, and because no effect has been observed upon the true or adrenalin sympathetic, physiologists have concluded that " there is no effect upon sympathetic nerves," and so have located the effect upon the plain muscle itself.

Schäfer writes (*Endocrine Organs*, p. 84): " The effect of the extract (pituitarin) upon the blood vessels is a direct one, and is not due, as in the case of the suprarenal hormone, to its stimulant action on sympathetic endings."

It is manifest that this amounts to saying that because the drug does not affect "*a*" it must therefore affect "*b*," and not "*c*" or "*d*" or "*e*," etc. The argument is fallacious, but is easily comprehended if it be assumed that the choice lies only between "*a*" (the true sympathetic) and "*b*" (the plain muscle itself). We have, however, already seen that good reason exists for believing that in the vagus or dilator sympathetic mechanism, which is activated by the depressor nerve, we have a possible alternative sympathetic mechanism, a possible "*c*" or "*d*" or "*e*" upon which drugs may conceivably act just as adrenalin acts upon the true sympathetic. Thus we are by no means forced to accept the direct-action-on-plain-muscle theory which, for us, is by no means the "only alternative."

The Action of Pituitarin corresponds in many respects to the Effect of stimulating the Depressor Nerve.—The fact that in experiments pituitarin has been found to raise blood pressure has closed the minds of investigators to the possibility of the drug having a depressor-like action. Certain facts concerning this pressor effect, however, merit very careful attention.

1. It occurs when a first injection of pituitarin is given, but it cannot be repeated by any subsequent injection. After the first injection the

effect of pituitarin is to cause a marked, sharp fall of blood pressure in the majority of instances. Schäfer (*Endocrine Organs*, p. 86, under Fig. 57) writes (Fig. 106 (after Schäfer)):—

> " The effect of the first injection is to produce a rapid rise of arterial pressure to a maximum followed by a gradual fall to the original level . . . the large oscillation of pressure in the pulsations on the fall of the curve are due to slowing of the pulse rate. The effect of the ' repeat ' injections is to cause in each case a marked fall followed by an after-rise."

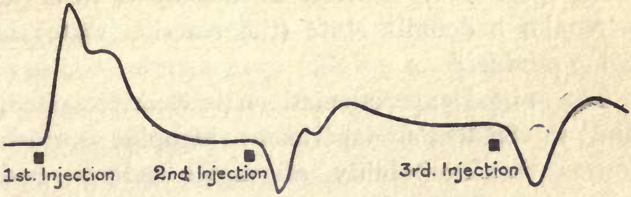


FIG. 106.

2. The action of pituitarin on other structures affected by it is not subject to the same reversal on repeating the dose. Biedl points out and Schäfer confirms that " on the heart's action the repeat dose has the same effect as the first dose." The same holds good for the effect on the uterus, bowels, etc. (The effect on the heart is " slowing with increase in force of the individual beats," which is an exact description of the vagus effect.)

3. The pressor effect of the first dose lasts much

longer than the depressor effects of subsequent doses.

These three facts—namely, that a first dose causes a rise of blood pressure which lasts some time, whereas other doses cause, as a rule, a fall (or no effect) which lasts a very short time, yet both doses have the same effect on heart, uterus, bowels, etc.—are impossible to explain unless we imagine, as we have good reason to, that during the first dose a definite " state " (the rest state) is being produced, exactly as during a dose of adrenalin a definite state (the reaction state) is being produced.

The animal experimented on is clearly excited, and in Schäfer's experiment atropine — which lowers the excitability of vagus endings—had been given to it. It was consequently under the influence of its sympathetic system—that is to say, it was in the reaction state, with its blood lakes tending to be closed and its muscles tending to be full of blood.

[The fact that atropine affects vagus endings by no means precludes the possibility of pituitarin acting upon those endings after atropinization. Captain Marris has shown that atropine in moderate doses fails to " release " the heart in typhoid fever; he explains this by suggesting that the vagus is powerfully stimulated in that fever (*i.e.*, atropine is powerless against the specific typhoid toxin). If pituitarin is the auto-drug of the dilator or vagus sympathetic action, then atropine in moderate doses will almost certainly fail against a dose of pituitarin just as it fails against a dose of typhoid toxin.]

Now let us suppose that upon this picture of the reaction state the rest state is suddenly induced by a drug having the effect of the vagus or dilator sympathetic—*i.e.*, opening the blood hearts and inducing active peristalsis in the vessel areas between those blood hearts—and so emptying the muscles of blood and bringing this mass of blood from the muscles back into the blood lakes. The inevitable result will be that, while the pressure in systemic arteries may fall for an instant as the tension of the reaction state is relaxed, *it will almost certainly rise again while the bulk of muscular blood surges through the arteries on its way back from the muscles* to the opened blood lakes. We know that pituitarin constricts all systemic vessels, and thus perceive that not only does it open the blood lakes, it also shuts the muscular blood field.

As has already been described, it is a matter of observation that after exercise the blood pressure falls and then rises again steeply, gradually declining thereafter to normal (Fig. 107). We have explained this second rise already in terms of blood-transference from the muscles back into the blood lakes or sphincters. It must never be forgotten that by taking blood pressure in or upon a systemic artery one takes it in or upon a passage-way of the blood stream. Any movement of blood, therefore, from one area to another will cause a crowding of the passage-ways, just as any movement of troops must cause a thronging and crowding of the lines of communication between one area and another.

The Interpretation of Blood Pressure.—When the reaction state begins, the ways are crowded as blood surges to the muscles: they remain crowded because the blood cisterns hold more blood than the muscles can contain, and for other reasons.

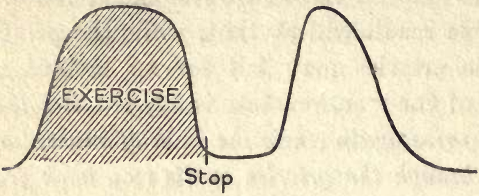


FIG. 107.

This is like troops going up to the front and being heavily supported by bodies of reserves behind. Then when the rest state begins the reserves are withdrawn suddenly, and for the moment the ways are empty until the men at the front begin to surge home again, once more, and for a short time, filling up the ways.

Pituitarin acts at first to bring blood surging back from the Muscles, and thus raises arterial pressure which gradually declines as the transference of blood is completed.—Schäfer says that "frequently a preliminary fall precedes the rise produced by the first dose."

This, by our showing, represents the opening of the blood lakes or hearts (or in our simile the withdrawal of reserves). Then comes the surge back from the muscles. This surge goes on until the muscular system has disgorged its massing of blood and the lakes have filled up.

The Picture now is the Rest State, which is a State of Stable Equilibrium : the Reaction State being a State of Unstable Equilibrium.—The picture at this point is the rest state or normal state. The blood lakes are full. The muscles have been emptied. The arterial passage-ways are at easy pressure.

Now another dose of the drug is given. At once the blood lakes are more widely dilated (just as a dose of adrenalin more sharply contracts them). Naturally and necessarily the pressure falls. As soon as the effect passes, the pendulum swings a little and the blood lake contracts to its normal again and a small rise follows. This is precisely what is seen during all subsequent injections of pituitarin after the first injection.

The Effect of Pituitarin on Vessels is just as rapid and sudden as the Effect of Adrenalin.—It has been stated that the effect of pituitarin on vessels lasts longer than that of adrenalin. This is true only of the pressor effect. It is not true of the depressor effect. But we have just seen that the pressor effect is only indirectly due to the pituitarin—that, in fact, it is really an effect of hydro-dynamics, a physical matter. The depressor effects, on the other hand, are directly due to the drug, and represent *actual openings* of the blood lakes.

This is more clearly understood when the preliminary fall of blood pressure often observed before the rise of pressure after the first dose is remembered. That preliminary fall represents the direct action of the drug in opening the blood lakes. The rise comes as the indirect consequence. Naturally if the muscles are very full of blood the rush back of blood from them to the opened blood lakes will be so sudden as to swallow up and obscure the initial fall. Only a pressor effect will be shown in the tracing.

The Effect of Pituitarin does not change upon any Structure.—We therefore perceive that the so-called difference between the first dose of pituitarin and subsequent doses is not a real difference at all. There is no difference between the effects. Only during the first dose the reaction state is changed into the rest state. In subsequent doses the rest state is intensified momentarily.

Vessels upon which Adrenalin has no effect or a Dilator effect are actively constricted by Pituitarin.—The loose statement that pituitarin constricts all systemic vessels is usually supported by two conspicuous instances, the pulmonary and coronary arteries. Now we saw that adrenalin would necessarily not act upon either of these structures for the reason that they are systemic vessels connecting blood lakes or sphincters—and adrenalin constricts only the blood lakes or sphincters.

But if pituitarin acts like the vagus or dilator sympathetic it will, by our showing, exercise its effect in activating these vessels connecting the blood lakes, *i.e.*, these peristaltic areas. We should, therefore, expect to find it "constricting" vessels to muscles and vessels between one blood lake or blood heart and another. And this we find it doing in point of fact.

Naturally this action on the vessels to muscles goes a long way to explaining the backward rush of blood from the muscles to the opened blood lakes, which, as we have just seen, accounts for the so-called pressor effect of pituitarin.

Schäfer writes: "Moreover, while adrenalin either produces no action on the coronary vessels, or, in some cases, causes them to dilate, the autacoid of the pituitary constricts them as it does most other systemic arterioles; this is true also for the pulmonary vessels."

The Effect of Pituitarin on the Bowels is exactly opposite to that of Adrenalin, and is exactly similar to that of the Vagus.—Adrenalin inhibits all peristalsis in the bowels, but at the same time constricts all the bowel sphincters. Pituitarin has the exact opposite effect—it stimulates peristalsis and opens sphincters: the vagus nerve has the same action. This action on this tissue was long ago defined and cleared up by Gaskell.

The Effect of Pituitarin on the Kidney affords a further clue to the Problem.—The effect of pituitarin on the kidney has interested every observer of the action of this drug. Before we discuss it we may recall the action of the depressor nerve on this organ :—

"*Ludwig and Cyon observed that on stimulating the depressor the kidney flushed red with blood*" (Schäfer, quoted above).

The effect of adrenalin on the kidney we have already discussed. It constricts that organ. Thus, in the reaction state, if the state is not so severe as entirely to shut the kidney, water is forced by main pressure out of the renal filter; if the state is so severe as to shut the organ, no water passes (adrenalin given intravenously causes stoppage of urine, given subcutaneously it causes increase of urine—pale watery urine).

The effect of pituitarin on the kidney is thus described by Schäfer (*Endocrine Organs*, p. 88):—

> "The renal arteries form an exception to the constrictor effect produced by pituitary extract, since they dilate as a consequence of the addition of the extract to the circulating blood, and dilatation is correlated with an increase of secretion of urine. . . . The increased secretion may be in part brought about by the increased flow of blood through the kidney vessels, due to the circumstance that they undergo dilatation while all the other systemic arteries are contracting . . . it may occur in the absence of any obvious arterial change . . . and the effect of a second and subsequent doses of the autacoid administered soon after the main result of the first dose has passed off is again

to produce an increase in the urine flow, although the blood pressure as the result of these after-doses does not rise, or falls instead of rising . . . the experiment clearly demonstrates that the autacoid affects not only the blood vessels of the organ but also its secreting cells. . . ."

The effect, then, is similar, so far as the vessels are concerned, to the effect of stimulation of the depressor nerve.

The fact that the renal arteries are dilated would lead us to suppose that they belong to the mechanism of the abdominal blood lake or sphincter. This is at once borne out by noting the effect of adrenalin on these structures and on the kidney itself—which is to constrict them (Fig. 108).

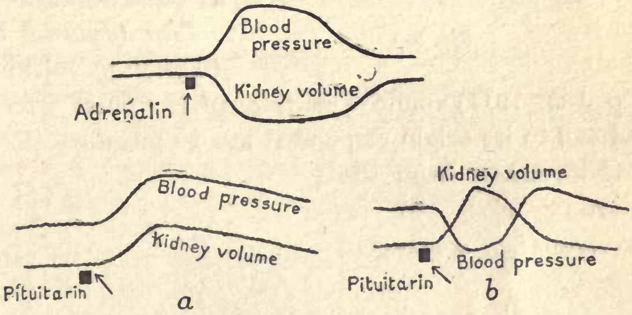


FIG. 108.

In the same figure we see the effect of pituitarin, which always dilates the kidney.

Are we therefore to suppose that the renal artery from the aortic end of it onwards belongs to the mechanism of the mesenteric blood lake and not, like other systemic arteries, to the peristaltic mechanism connecting the blood lakes?

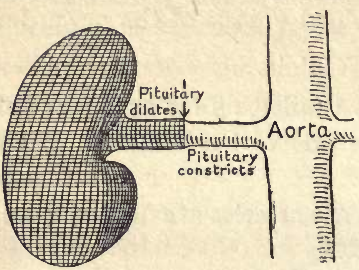


FIG. 109.

Some exceedingly interesting experiments have been carried out on this artery, which show that the assumption would not be quite accurate.

Cow (*Journal of Physiology*, vol. xlii. p. 135, 1911) found that rings of the renal artery *distal* to its origin responded less to pituitary than those taken from the artery close to its origin (*i.e.*, contracted less).

In Pal's experiment the distal rings were found definitely to relax.

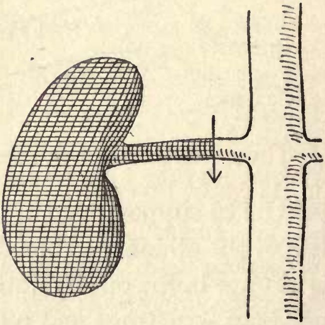


FIG. 110.

These experiments might indicate that the renal artery actually marks the transition from a connecting, peristaltic area to a blood lake or sphincter area, and that the position of the arrow in Figs. 109 and 110 shows roughly where the systemic vessel ends and the "sphincter of the kidney"—which is, of course, part of the abdominal blood lake or sphincter—begins. We thus observe that the effect of pituitarin on the kidney is diametrically opposed to that of adrenalin.

Summary of Action of Pituitarin.—Thus we perceive that the main actions of pituitarin bear the most striking likeness to those of the vagus or dilator sympathetic as revealed by stimulation of the depressor.

These may be set down in tabular form thus :—

Vagus or Dilator Sympathetic.	**Pituitarin.**
1. Slows heart.	1. Slows heart.
2. Lowers blood pressure.	2. Lowers blood pressure after first dose (the effect of first dose is quite explicable, as we have seen).
3. Increases peristalsis.	3. Increases peristalsis.
4. Flushes kidney with blood.	4. Flushes kidney with blood.
5. Constricts bronchioles.	5. Constricts bronchioles.

The comparison with adrenalin is no less striking in its exact reversal :—

Adrenalin.	**Pituitarin.**
1. Quickens heart.	1. Slows heart.
2. Raises blood pressure sharply.	2. Lowers blood pressure sharply after first dose.
3. Inhibits peristalsis.	3. Increases peristalsis.
4. Contracts kidney.	4. Dilates kidney.
5. Opens bronchioles.	5. Constricts bronchioles.

There the matter must be left pending further investigation.

Note.—We may add that recent work (Cannon, Levy, and others) has gone a long way to show that while thyroid always augments the pressor action of adrenalin, the hormone of the pancreas (and possibly that of the parathyroid body) tends to augment the dilator or rest picture, and to oppose the adrenalin picture. A wide field of investigation evidently lies here, and it may be that a relationship between these endocrines and the sympathetic or vagus-dilator systems as precise as that established between adrenalin and the sympathetic system will be made out.

CHAPTER XV

THE RELATIONSHIP BETWEEN THE REST STATE AND THE REACTION STATE

WE have been able to observe that the reaction state is invariably followed by the rest state, and also that sudden production of the rest state or dilator state in excess leads at once thereafter to the reaction state.

There is a Pendulum Balance between the Rest State and the Reaction State.—In other words, to induce the reaction state is not only to induce that state, but is also to store up, as it were, the rest state mechanism so that as the reaction state wanes the rest state pulls in, and will swing the pendulum further to the rest state side.

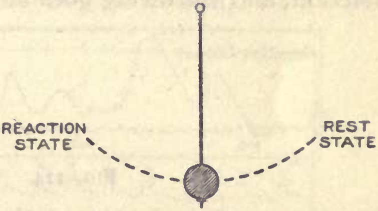


FIG. 111.

This idea is illustrated in Fig. 111.

It is also illustrated in Fig. 112 and Fig. 113. In Fig. 112 (and also in Fig. 114) the patient is in the reaction state, his temperature being 101° F. Inspiration by weakening the reaction state and relaxing the abdominal wall at once dilates his blood lakes and slows his pulse, as is well seen. The result, however, is an immediate following vaso-constriction, and the swing goes on for some time. In Fig. 113, on the other hand, the patient's temperature is 97·2° F., and he is in the rest state (a very weak man). Inspiration in his case has a marked vaso-constricting effect (it will be remembered

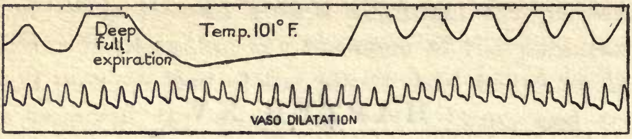


FIG. 112.

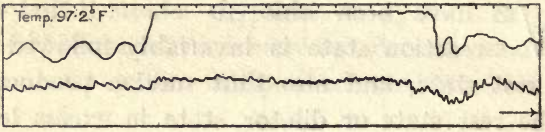


FIG. 113.

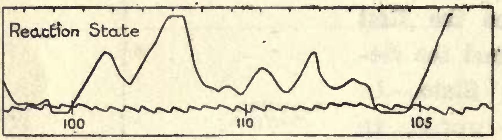


FIG. 114.

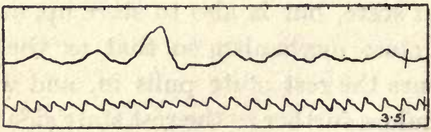


FIG. 115.

that we saw that in the rest state inspiration was the active phase, while in the reaction state it was the passive or negative phase) and the heart is quickened. No sooner, however, is the inspiratory effect ended than at once the opposite picture is induced, with vaso-dilatation and slowing. Here again a wave movement continues thereafter. In Fig. 115 another illustration of the condition observed in Fig. 113 is seen. This latter condition is spoken of (Fig. 114) as "sinus arrhythmia" (slow pulse at expiration: quick pulse at inspiration). So that *in the reaction state the "sinus arrhythmia" is reversed.* Figs. 116 and 117 show also wave motions in the pulse met with during disturbed balance periods.

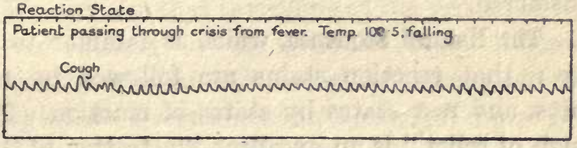

Reaction State
Patient passing through crisis from fever. Temp. 100·5, falling
Cough

FIG. 116.

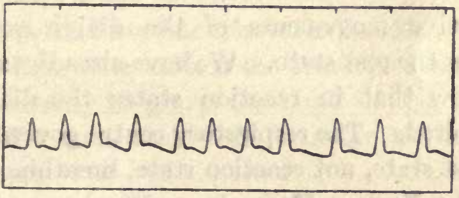


FIG. 117.

We shall prefer to consider these phenomena generally as representing the swing of the pendulum.

The Links between the Rest State and the Reaction State.—There are many links between the rest state and the reaction state, and these may now be considered.

1. **The Natural Sequence,** which is familiar to all men: that reaction states are followed by rest states, and rest states by states of reaction. The "sigh of relief" is an excellent illustration of this, so is the expression "breathing oneself."

2. **The Respiratory Mechanism.**—We have already described the two types of breathing in great detail, and need not recapitulate. Enough that the entire lack of oxygenation occurring during active reaction states, by raising the acid content of the blood to an intolerable limit, soon induces the active movements of the diaphragm which usher in the rest state. We have already explained our view that in reaction states the diaphragm is inhibited. The respiratory centre governs therefore rest state, not reaction state, breathing.

3. **The Nervous Mechanism.**—We have observed that the depressor nerve is so situated in the aorta that stimulation of it will be effected by a high aortic pressure—that is to say, the depressor nerve will excite the vagus or dilator sympathetic whenever the reaction state becomes too extreme and pressure in the arteries is in consequence raised too high. Thus the rest state will be ushered in.

4. The Auto-Drug Mechanism.—We saw that from its position the pituitary gland would be likely to be compressed when and if cerebral pressure was continued so as to fill the venous cisterns of the cranium to overflowing.

We saw also that this filling of the venous cisterns is an activity of the reaction state—adrenalin sympathetic — so that when the *adrenalin sympathetic becomes over-active the pituitary gland is activated* and the chemical pituitarin cast out. In this way, we suggest, the adrenalin sympathetic picture is modified by the pituitarin sympathetic picture or vagus-dilator sympathetic picture.

We have thus a physical, a respiratory, a nervous, and a neuro-chemical mechanism whereby the reaction state is tempered and controlled by the rest state.

The Reaction State thus induces the Rest State directly; and the Rest State induces the Reaction State.—It is very necessary to grasp the truth of this proposition. We shall deal with it under the headings just given.

1. The Natural Sequence.—Exercise leads to fatigue inevitably; rest leads to activity.

2. The Respiratory Mechanism.—The respiratory centre, having been stimulated with CO_2 and other products of reaction, powerfully stimulates the diaphragm, which in the rest state oxygenates the blood by deep and full breathing.

The result of this is that the respiratory centre becomes less and less irritable as the blood becomes better and better oxygenated. At last the respiratory centre becomes unirritable and the diaphragm stops of its own accord in an apnoeic period.

Manifestly the way has now been prepared for the reaction state to begin again if necessary (the man has "breathed himself").

3. **The Nervous Mechanism.**—The depressor nerve is activated by high pressure in the aorta. It has recently been shown that low pressure in the aorta will activate the sympathetic system and cause adrenalin to appear in the blood stream in increased amount.

4. **The Auto-drug Mechanism.**—As soon as the rest state is induced the pressure in the venous sinuses will be relieved and the pituitary gland will cease to be squeezed. This will remove the opposition to adrenal activity.

The Vagus-Dilator Sympathetic or Pituitarin Sympathetic is, however, more than a mere governor on an engine. It is the boiler in which the steam is generated also.—There can be no doubt concerning the tempering effect of the rest state (vagus or pituitarin sympathetic) upon the reaction state. But it would be a mistake to view this as the whole truth concerning the rest state. For not only is the rest state a state of rest: *it is also a state of replenishment.*

The marked increase of peristalsis and the secretory effects which follow vague stimulation are a definite lead in this direction. The effect of pituitarin in causing a true diuresis (as opposed to a mere pressure diuresis), and also its effects on glands like the mammary gland, furnish further suggestive material of thought.

Clearly in the rest state the wear and tear and waste of the reaction states are made good.

1. The blood is cleansed of CO_2 and oxygenated.

2. The open peripheral sphincter or blood lake allows of cooling of the blood; and the sweating which tends to occur after the reaction state or during it, as it becomes tempered by the rest state, also helps to induce cooling.

3. The induced peristalsis and flow of digestive and other juices (which can be produced by vagus stimulation) promotes appetite ("hunger pains") and digestion.

4. The excretory mechanism, especially the kidney, is stimulated to true glandular action, and the waste products of effort got rid of.

5. The pressure in the venous chamber of Keith being now fallen, the thoracic duct is no longer shut (by the valve at its orifice), and so the chyle can flow again into the blood.

This mechanism of replenishment necessarily produces a fullness which like a sensitized photographic plate awaits exposure. The man has, so to speak, "got steam up." Moreover, the governor action of the vagus or dilator sympathetic is withdrawn—there being nothing to govern. He is ready for the unloosing of his powers in reaction.

The Reaction State always dominates the Rest State.—This is a truism of life. Experimentally it has been shown that in a dog with a gastric fistula the slightest excitement caused an instantaneous stoppage of peristalsis and gastric juice flow. The mere sight of a cat in the distance was enough to inhibit entirely the rest state picture.

In certain states of ill-health the Rest State may follow the Reaction State with such violence as to produce Collapse.—This is seen in fainting (*vide* above, Lewis's description of a faint in a soldier with disordered action of the heart), and tends, as might be expected, to be most common in a situation in which sensory stimuli are reduced in amount, *e.g.*, in a hot, "airless" room. The modern discovery that it is not oxygen which counts in ventilation, but currents of air, is interesting in this respect.

Health is a balance between the Reaction State and the Rest State.—In the case of a healthy man the rest state and the reaction state are so well balanced that every act can be accomplished with the minimum of waste and expenditure. As he runs, for example, his "second wind" comes to him without loss of his "first wind"—the rest state gently regulates the reaction state, toning and tempering it, yet not in any sense interfering with its efficacy. His skin gradually opens (the peripheral sphincter or blood lake gradually opens) to receive and cool the heated blood and to reduce the " head of steam " to a nicely calculated sufficiency (just as a careful motor driver reduces gas and gives more air when the engine has got well " run in " on a journey).

Moreover, the more accustomed the task the less effort is required to carry it out, for the nervous system has of itself learned the balance required, and can and does adjust rest to reaction in such a way that, while the tyro groans and sweats, the master mechanic shows no symptoms of effort at all.

CHAPTER XVI

ACUTE DISEASE AND THE NERVOUS SYSTEM

THERE is probably no department of medicine which stands so urgently in need of investigation as that comprising the acute febrile diseases. Already signs are not wanting that these diseases will fully repay any pains that may be spent upon them . . . the most notable of these signs being the recent discovery by Captain Fairly Marris that in typhoid fever atropine fails to release the pulse.

Captain Marris has suggested, as a possible explanation of this phenomenon, that in typhoid the vagus is stimulated to increased activity, and the generally slow pulse, the low blood pressure usually encourtered, the peristalsis, and the lack of "life" and energy—the *τῦφος*—(*i.e.*, failure to enter the reaction state) lend colour to that view.

If this line of thought is justified, the possibility that the Toxins of acute diseases act like Drugs must be considered.—This necessarily follows, and indeed a moment's consideration will show that the so-called "symptoms" of acute disease are not far removed from the "symptoms" met with in the varying activities of ordinary life.

Are all "Symptoms" of acute disease referable to the Reaction State, or the Rest State, or Modifications of them?—This is a matter of observation: it being a question of watching a great many patients in different illnesses of an acute kind. So far as the writer's observations go, he is of opinion that acute diseases do not "*produce symptoms*" at all in the commonly understood sense—that is to say, it is not the disease which produces the symptoms: it is *life* which produces them. The disease merely prepares the photographic plate. Life takes the photograph.

The case of the strychninised frog should be borne in mind in this connection. So long as the frog is kept perfectly still and unstimulated, no convulsions occur: but once touch it, it will go into convulsions. The strychnine, therefore, has prepared the nervous system of the frog by exciting it to a great degree, so that a light touch, which in ordinary conditions would have produced in the frog a light reaction state, now produces the most violent of all reaction states—a convulsion.

The patient with tetanus, who goes into a convulsion at the sound of a footfall, is another case in point. It is not the tetanus which produces the convulsion; it is the sound of the footfall. The tetanus prepares the photographic plate.

And a moment's consideration will show that a tetanic spasm, with the retracted head, the chest forcibly held open, the tense abdomen, etc., is an exact picture of the reaction state as we have described it—an exaggerated but an exact picure. Other disease pictures of the reaction state suggest themselves—cerebro-spinal fever, for example, the rigor of malaria, and so on.

A permanently over-excited Nervous System by laying the Patient open to excessive Reaction State Pictures on Slight Provocation must lead to Chronic Disease. —If we imagine the case of a man one or other of whose sympathetic mechanisms is permanently hyper-excitable (as the result, say, of a chronic infection) we can easily understand that this man will be doomed to react excessively to all the stimuli of life, and will, therefore, while young, be regarded as a "functional" case, and when older, probably as an organic case—the nature of his organic disease *depending in all probability upon which of his organs plays out first under the ruinous terms on which he is forced to live.*

This man is going to pass deeply into the reaction state or the rest state on the slightest provocation, because his nervous mechanism is permanently over-excited, or, in current phrase, because his "threshold of resistance is permanently lowered." Constant high-blood pressure on slight provocation, for example, or constant reaction or effort breathing, constant wear and tear and expenditure and waste will be his lot. It is easy to see such conditions as arterio-sclerosis and the other scleroses looming ahead of him.

This, briefly, is the physiological doctrine of disease; it merits the most earnest and patient investigation.

The writer's task is finished. If his work helps to stimulate an interest in the great processes of physiology which, being exaggerated or deranged, we call "disease," he will feel himself most amply rewarded.

INDEX

Abdomen, shape of, as indication of reaction state, 43, 44
Abdominal blood lake or cistern:
  emptying of, 48, 74
  in relation to heart, 85, 114, 116
  pulsations of, differentiated from aortic beats, 116
  pulsations of, differentiated from reversed heart beats, 118
Abdominal muscles:
  inhibited in rest breathing, 57, 59
  manner of contraction of:
    during mental effort, 37
    in reaction states, 24, 25, 26, 27
  viscera as piston of syringe in reaction states, 22
  wall, function of, in reaction states, 19, 20, 21
Acidosis, physiological, in reaction states, 33
Acute disease and the nervous system, 170 et seq.
Adrenalin:
  action of, 71 et seq.
  action of, in filling muscles with blood, 81
  action of, opposed by vagus, 127
  as adjuvant in emptying blood lakes during reaction states, 73
  compared with pituitarin, 159
  effect of, on arterial and venous pressure, 81, 82
  effect of, on blood pressure, 76, 129
Adrenalin:
  effect of, on bronchioles, 76
  effect of, on heart rate, 74
  effect of, on kidneys and urine flow, 82
  effect of, on muscular vessels, 80
  effect of, on peristalsis, 74
  poured out in reaction states, 70, 72, 83
  the drug of the reaction states, 83
Adrenalin sympathetic:
  effect of pituitarin on, 147
  opposed by vagus depressor sympathetic, 131
Air:
  as a pneumatic buffer between blood and thrust of abdominal muscles, 31
  in bowels as a pneumatic buffer, 34, 69
  output of, in reaction states acts as a regulator of blood output, 30
  prevented from leaving thorax in reaction states, 29, 30
  sucked into thorax in rest state, 60
Anacrotic waves:
  as a demonstration of peristalsis in arteries, 106
  explanation of, 106
Anatomy of abdomen in relation to the reaction state, 43
Anger, pallor of, 136 et seq.
Aortic beats, differentiated fro beats of abdominal blood lake, 116
Apocodeine, effect on pulse of, 103

Arteries, systemic, as peristaltic areas like the peristaltic areas of bowel, 95
Arterioles of periphery, mesentery and lungs are hearts, 111
Atropine, action of, on vagus endings, 150

Barrel shape of thorax changed into funnel shape in reaction states, 21
Bayliss, Professor, Preface, 80, 92
Blood:
  disposal of, in reaction states, 33, 34
  forced out of thorax in reaction states, 30
  oxygenation of, stopped in reaction states, 32
Blood lakes:
  effect of adrenalin on, 73, 76, 77
  embryological relationship to heart, 88
  in reaction states and rest states, 58
  relation of, to heart, 88 et seq.
  relation of, to systemic arteries, 107
Blood pressure:
  affected by descent of diaphragm in rest state, 60
  effect of adrenalin on, 76, 129
  effect of pituitarin on, 149, 150, 151
  high, as excitant of depressor system, 130
  in reaction states and rest states, 58, 59, 151
  interpretation of, 152 et seq.
Blood wave passing from blood lakes to muscles in reaction state, 48, 49
Blush, explanation of, 136
Blushing, in rest state, mechanism of, 140
Bowels, effect of pituitarin on, 155
Brain:
  emptied of blood during rest breathing, 58
Brain:
  flushed with blood in reaction, 34, 38, 39, 63 et seq.
Breath, held during effort, 41
Bronchioles:
  action of vagus on, 129
  effect of adrenalin on, 76, 129
Bulbo-sacral sympathetic system, 127

Canon, Professor, 93, 160
Cerebral cavity as blood cistern in reaction state, 34, 63 et seq.
Cerebro-spinal fluid in reaction state, 64
Chest, shape of, in reaction state, 22
  shape of, in rest state, 56
Choline, effect of, 146
Chronic disease as result of permanent over-excitability of nervous system, 172
Collapse, mechanism of, 168
Coronary arteries, effect of pituitarin on, 155
Costal margins:
  as pivot point of expiration in reaction states, 14
  in reaction states, 11
Coughing as a form of reaction, 28
Cow, Dr., on the renal arteries, 158
Cranium:
  events in, during reaction states, 63 et seq.
  venous pressure in reaction states, 64

Deltoid muscle in reaction states, 10, 21
Depressor nerve:
  action of, 129, 130
  action of, on kidney, 130, 156
  key to vagus depressor system, 129
  link between reaction and rest states, 130, 131
  pituitarin in relation to, 130, 131

Diaphragm :
  inhibited in reaction breathing, 17
  muscle of rest breathing, 57, 60
Diastole, vagus the nerve of, 127 et seq.
Dicrotic wave :
    explanation of, 101
    in reaction states, 102
    in rest states, 102
Dilator sympathetic system, 128
Dixon and Halliburton, *Journal of Physiology*, on cerebrospinal fluid, 64
Ductless glands :
    and the reaction state, 68 et seq.
    grouped according to phenomena of reaction, 68

Expiration, active phase of reaction breathing, 20
  in reaction breathing, mechanism of, 13
  in rest state, the passive phase, 59
Expiratory periods longer in rest breathing, 57

Facial changes in reaction and rest states, 137, 138, 139
Fainting :
  as a rest state phenomenon, 67, 141, 142, 168
  pallor of, explanation of, 136
Fevers and the nervous system, 170 et seq.
Flushing :
  in the reaction state, 138
  of anger and shame differentiated, 136, 137
Fraser, Dr. F. R., on adrenalin, 71
Funnel-shaped thorax in reaction, 21

Gaseous exchange stopped in reaction states, 55
Gaskell, *Involuntary Nervous System*, 78, 80, 88, 107
Gasp, the, in reaction states, 3, 16
Glottis :
  closing of, in reaction states, 19, 20, 29, 30
  in mental effort, 37
Gunn and Chevasse, effect of adrenalin on superficial veins, 77

Health, a balance between reaction states and rest states, 168
Hearing, as a form of reaction, 38
Heart :
  action on, of adrenalin opposed by vagus, 127
  as a sphincter muscle, 88
  beats of, differentiated from beating of mesenteric or abdominal blood lake, 118
  beats of, differentiated from beating of pulmonary blood lake, 121
  effect of adrenalin on rate of, 74
  embryological relationship of blood lakes to, 88
  in "plus-systole," 85
  in reaction states, 35
  not two hearts, but five, 90
  peripheral blood lake is a sphincter beating synchronously with, 99
  relation of, to blood lakes, 85 et seq.
  relation of, to vasomotor system, 90
Hearts, the :
    activated in reaction states, 113
    inhibited in rest states, 113
    mechanism of arterioles and, 108, 112
    peristalsis of arteries and, 107

Inferior thyroid vein, 52
Inspiration :
  in reaction breathing, mechanism of, 15
  in rest breathing caused by descent of diaphragm, 57
  in rest states, the active phase of respiration, 59

Inspiratory periods shorter in rest breathing, 57
Intestine, law of the, 92 et seq.
Intracranial pressure in reaction states, 65

Keith, Professor:
  on peristalsis in bowels, 92, 93
  venous cistern of, 49, 58
Kidney:
  effect of adrenalin on, 82
  effect of pituitarin on, 155, 156
  effect of thrust of abdominal wall on, 45, 53

Langley, Professor, on adrenalin, 72
Lewis, Dr., on soldier's heart, 141, 147
Liver:
  contents in relation to venous cistern of Keith, 50
  effect of thrust of abdominal muscles on, 45
  flushed by portal blood during reaction states, 49
Luciani, Professor, *Physiology*, 75, 107, 127
Lungs, manner in which blood is driven out of them in reaction states, 32, 33

Mackenzie, Sir James, Preface
Markings on organs, in reaction states, 43
Marris, Captain:
  on atropine test, 150, 170
  on venous pressure, 82
Mental effort as producer of reaction states, 37
Mesenteric blood lake:
  differentiation of beats from aortic beats, 116
  differentiation of beats from heart beats, 118
  emptied in reaction states, 46
  in relation to heart, 85, 114
  tracings of action of, 114
Mott, Dr., on cerebro-spinal fluid, 64
Movements of thorax, effect of retraction of head upon, 7
Muscles:
  as blood cisterns in reaction state, 34
  blood supply of, and pituitarin, 152, 155
  in rest breathing, 56, 57
  venous blood dammed back in, in reaction states, 51
Muscular mechanism in reaction states, extent of, 17
  picture in starting, 6

Nasal tracings showing beats of pulmonary blood lake, 121, 122
Negative pressure in systole, 125
Nerve of diastole, vagus as, 127 et seq.
Nervous system:
  and acute disease, 170 et seq.
  permanent over-excitability of leading to chronic disease, 172
Nutrition, vagus-depressor system as controller of, 164

Organs, markings on, in reaction states, 43
Oxygenation of blood stopped in reaction, 32

Pallor:
  in the reaction state, 34, 137
  in the rest state, 141
  of anger differentiated from that of fainting, 136
Pancreas:
  in the reaction state, 51, 68, 69, 160
Pancreatic blood conveyed to muscles in venous chamber of Keith, 51
Pectoralis major muscle in reaction states, 10, 21
  minor muscle in reaction states, 12
Peripheral blood lake:
  beating of, cannot be shown in a plethysmogram, 143

Peripheral blood lake:
    emptied by adrenalin, 76
    in relation to heart, 85
    is a sphincter beating synchronously with heart, 99
Peristalsis:
  effect of adrenalin on, 74, 95
  effect of vagus on, 95
Peristalsis of arteries:
    activated by vagus-depressor system, 132
    a different mechanism from systole and diastole of sphincters or hearts, 107, 113
    anacrotic pulse as proof of, 106
    augmented in rest state and inhibited in reaction state, 133
    is a continuous rhythm, 113
    physiological findings supporting, 107
    proofs of, 100 et seq.
Peristaltic areas in bowel—analogous peristaltic areas in systemic arteries, 95
Physiological doctrine of disease, 173
Piston of syringe, abdominal viscera as in reaction states, 22
Pituitarin:
  action of, 144 et seq.
  compared with adrenalin, 159
  depressor effects of, 149, 150, 151
  effect of, is constant, 154
  effect of, like effect of vagus-depressor system, 145, 148, 159
  effect of, on blood pressure, 149 et seq.
  effect of, on bowel, 155
  effect of, on coronary arteries, 155
  effect of, on kidney, 155
  effect of, on muscular blood supply, 152, 155
  effect of, on pulmonary vessels, 155
Pituitarin:
  effect of, on renal arteries, 155
  effect of, on vessels compared to that of adrenalin, 153
  effect of, on vessels not affected by adrenalin, 154, 155
  effect of, on vessels to muscles, 155
  pressor effect of, explained, 149
Pituitary body, squeezed in reaction state, 65 et seq.
"Plus-systole" of heart and blood lakes, 85
Portal blood, flushing out liver in reaction states, 49
  vein, engorged in reaction, 46
Pulmonary blood lake:
    differentiation of beats of, from heart beats, 121
    effect of pituitarin on, 155
    emptied by adrenalin, 77
    in relation to the heart, 87
Pulse:
  anacrotic, 106
  effect of apocodeine on, 103
  effect of vaso-dilators on, 102, 103
  experimental production of, 100
  explanation of, 96
  in relation to vasomotor phenomena, 96 et seq.
  quickened in inspiration in rest state, 60
  types of, in rest and reaction, 102, 103 et seq., 133
Pylorus, closed in reaction states, 47

Rapport, Dr., work on blood pressure, 58
Reaction breathing, mechanism of, 12
Reaction state, the:
    abdominal blood cistern or lake, emptying of, in, 48 et seq.
    abdominal muscles, manner of contraction of, in, 24, 25, 26, 27
    abdominal viscera as piston of syringe in, 22

Reaction state, the :
- abdominal wall, functions of, in, 19, 20, 21
- acidosis, physiological, in, 33
- adrenalin the drug of, 83
- air output in, acts as regulator of blood output, 30
- air prevented from leaving thorax in, 29, 30
- anatomy of abdomen in relation to, 43 et seq.
- as accompaniment of all effort, 37 et seq.
- as excitant of rest state, 66, 67, 161 et seq.
- barrel-shaped chest changed to funnel-shaped chest in, 21
- blood, disposal of, in, 33, 34
- blood forced out of thorax in, 30
- blood oxygenation stopped in, 32
- blood wave from lakes to muscles in, 48, 49
- breath held in, 39, 40, 41 et seq.
- cerebral cavity as blood cistern in, 34
- cerebro-spinal fluid in, 64
- compared with rest state as regards breathing, 57
- costal margins as a pivot of expiration in, 11, 14
- cough as a form of, 28
- deltoid muscle in, 10, 21
- diaphragm, inhibited in, 17
- dicrotic waves in, 102
- dominates rest state, 168
- ductless glands in, 68 et seq.
- effect of adrenalin in, 71 et seq., 75
- events in cranial cavity during, 63
- excitation of depressor nerve in, 130, 164
- expiration as active phase of, 20
- expiration, mechanism of, in, 13

Reaction state, the :
- failure to achieve is failure to live, 91.
- flushing in, 138
- funnel-shaped thorax in, 21
- gaseous exchange in, 55
- gasp in, 3, 16
- glottis, closing of, in, 19, 20, 29, 30
- hearing, seeing, speaking as forms of, 38, 39
- heart in, 35
- inspiration in, mechanism of, 15
- intracranial pressure in, 65
- kidney in, 45
- liver in, 45
- lungs, manner in which blood is driven out of them in, 32, 33
- markings on organs in, 43
- mental effort as producer of, 37
- muscles as a blood cistern in, 34
- muscular mechanism in, 17
- pallor in, 34, 137
- pancreas in, 51, 160
- pectoralis major muscle in, 10, 21
- pectoralis minor muscle in, 12
- peristalsis of vessels inhibited in, 113, 133
- piston of syringe, abdominal vessels as in, 22
- pituitarin as opponent of, 153
- pituitary body in, 65
- portal blood flushing out liver in, 49
- portal vein in, 46
- pulse in, 102, 133
- pylorus in, 47
- respiratory centre in, 66
- scapula as muscular pivot in, 9
- serratus magnus muscle in, 10, 11, 21
- shape of abdominal cavity as indication of, 43
- sheath of rectus, mechanism of, in, 26

Reaction state, the:
  shoulder as pivot point in, 10
  spleen in, 45
  sterno-mastoid muscle in, 8
  stomach in, 45, 47
  suprarenals in, 45, 68
  tendinous intersections of recti muscle in, 26
  thoracic duct in, 54
  thorax, emptied by abdominal muscles in, 20
  thyroid in, 52, 68
  trapezius muscle in, 6, 7
  urine in, 53
  vasomotor system in, 91
  venous chamber of Keith in, 50
  venous return from periphery prevented in, 51
  vomiting in, 47
Recti muscles in reaction, 14, 26, 27, 28, 29 et seq., 44
Renal arteries:
  effect of pituitarin on, 156
  physiology of, 158
Respiration, effect of adrenalin on, 75
Respiratory centre:
  excited in long-continued reaction, 66
  governs rest state, 164
Rest state:
  abdominal muscles in, 57
  blushing in, 140
  diaphragm in, 57
  dicrotic waves in, 102
  effect of, opposite to that of reaction, 57
  exaggerated after reaction state, 62
  excites to production of reaction, 66, 67, 161 et seq.
  inspiratory and expiratory periods in, 57, 59
  muscles in, 56
  pallor in, 141
  peristalsis of vessels in, 113, 133
  pituitarin as auto-drug of, 153
  pulse in, 60, 102, 133
Rest state:
  respiratory centre in, 164
  state of depressor and vagus activity, 131
  suprarenals in, 61
  venous cistern of Keith in, 58
  viscera in, 57
Retraction of head in starting, 6, 7
Return to normal after starting, 5

Salathe, experiment on hutch rabbit, 73
Scapula, as a pivot point in reaction states, 9
Schäfer, Professor, 73, 75, 81, 83, 88, 110, 111, 129, 147, 149, 152, 155
Seeing, as a form of reaction, 38
Serratus magnus muscle in reaction, 10, 11, 21
Sheath of rectus, mechanism of, in reaction, 26
Sherrington, Professor, 84
Shoulder as pivot in reaction states, 10
Sigh of relief in rest state, 56
Simple functions, study of, 2
Sinus arrhythmia reversed in reaction states, 163
Skin, blood lake of, emptied by adrenalin, 77
Speaking as a form of reaction, 38
Sphincter, the heart as a, 88
Sphincters of bowel, 94
  action of adrenalin on, 95
  action of dilators on, 131
  action of vagus on, 95, 131
Splanchnic blood lake (*see* abdominal mesenteric blood lakes), 74
Spleen, effect of thrust of abdominal muscles on, 45
Starling, Professor, 92
Start, mechanism of a, 2, 3
Sterno-mastoid muscle in reaction states, 8
Subcutaneous plexus in relation to flushing, 139, 140

Subpapillary plexus in relation to flushing, 139, 140
Suprarenal blood conveyed to muscles by venous chamber of Keith, 51
Suprarenal capsules:
  effect of thrust of abdominal wall on, 45, 68
  in rest state, 61
Sympathetic, true:
  and adrenalin, 72, 83
  opposed by action of pituitarin, 147
  opposed by action of vagus-depressor system, 131
Sympathetic, vago-sacral or bulbo-sacral, 127 et seq.

Tendinous intersections of recti muscles in reaction, 26
Thoracic duct shut in reaction states, 54
Thorax:
  effect of thrust of abdominal muscles on, 29 et seq.
  emptied by abdominal muscles in reaction, 20
  in rest states, 57
Thyroid, augmenter of adrenalin action, 52, 68, 160
Toxins, action of, like drugs, 170
Trapezius muscle, in reaction states, 6, 7
Typhoid toxin on vagus, 150, 170

Urine:
  driven out of kidneys in reaction states, 53
  flow, effect of adrenalin on, 82

Vago-sacral sympathetic system, 127
  is a dilator system, 128
  opposes the true or adrenalin sympathetic, 131
Vagus:
  action on bronchioles, 129
  as a branch of depressor system, 129
  nerve of diastole, 127 et seq.
  opposes action of adrenalin on heart, 127, 128, 129
  supply of, to bowel reaction on bowel, 95, 129
Vagus-depressor system:
  and pituitarin, 144 et seq.
  as controller of nutrition, 166
Vaso-dilatation, effect on pulse of, 103
Vaso-formative cells of Schäfer, 88, 89
Vasomotor system:
  and pulse wave, 96, 97 et seq.
  in relation to heart, 90
Veins, deep, effect of adrenalin on pressure in, 81
  superficial effect of adrenalin on pressure in, 82
Venous cistern or chamber of Keith, 49
  as a means of bringing portal blood to muscles, 50
  in rest states, 58
  return from periphery stopped in reaction, 51
Viscera, descent of, in rest breathing, 57
Vomiting in reaction states, 47

14 DAY USE
RETURN TO DESK FROM WHICH BORROWED

Biology Library

This book is due on the last date stamped below, or
on the date to which renewed.
Renewed books are subject to immediate recall.

AUG 15 1959
AUG 8 1959

LD 21-50m-8,'57
(C8481s10)476

General Library
University of California
Berkeley

k

YB 79533